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THE USE OF ESTROGENIC SUBSTANCES IN ATROPHIC RHINITIS.*

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Estrogenic substances were first used in cases of atrophic rhinitis in the out-patient department of the Eye and Ear Infirmary on Aug. 1, 1937. At that time estrin was used more or less empirically, with the hope that results in the nose might be obtained, similar to those resulting from the work of Davis and Lewis in the vagina in cases of atrophic, or senile vaginitis, and gonorrheal vaginitis, respectively. A preliminary report of this work was given last November, in which the similarity in the pathology between atrophic vaginitis and atrophic rhinitis was pointed out, as well as certain physiological disturbances, notably menstrual abnormalities, which were encountered rather frequently in patients with atrophic rhinitis.

This paper, a follow-up of the previous one, is not a complete scientific study of the problem, for although the results have been gratifying, the methods used thus far have been incomplete, and in many respects quite unscientific. For example, relatively few biopsies have been obtained, no endocrine studies have been done and, what is more important, no estrin determinations in urine specimens have been carried out. These latter determinations have not been done, because a reliable method of procedure has not yet been found, and also

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because no apparatus at present is available for carrying out such procedures.

I wish to review rather briefly some of the facts which have been discovered in the past few years concerning estrin. The follicular hormone (estrin, folliculin) can be obtained from the follicular liquid of man and various mammals. Allen and Doisey first found it in the follicular liquid of cows and sows. About 0.25 to 0.5 cc. of the liquid contains one mouse unit, which is the quantity producing estrus when injected into a castrated mouse. The human follicle just before it ruptures contains approximately 2 to 3 cc. of liquid. The hormone can also be demonstrated in the wall of the follicle shortly before it matures, and hormone production thus depends for its proliferation on the follicular apparatus. The substance is not formed uniformly nor constantly, but in cycles. After each menstruation it falls to a minimum but never entirely ceases, and, according to Zondek, attains its maximum in the pre-pregnant stage.

The hormone is found also in placenta. Recent investigations reveal that the estrus hormone is present in every human urine in small amounts. Zondek states that "the supposition that it might be derived from the food has proven untenable; the authors, therefore, assume that besides the sex glands and the placenta, there must be some other (extra-gonadal) source of estrin. The excretion of estrin increases about twenty-fold in women after attaining sexual maturity: in the postmenstrual phase about 50 mouse units, in the intermenstrual phase about 300 mouse units are found in a litre of urine."

It is a well known fact that atrophic rhinitis usually begins about the age of puberty, and lasts until the patient reaches 45 or 50 years of age, at which time it seems to get better spontaneously. This coincides with the increased production and excretion of estrin occurring in the so-called polyhormonal disturbances of women (polyhormonal amenorrhea), menorrhagia, the first stage of the climacteric.

As soon as pregnancy occurs and the fertilized ovum has come into contact with the maternal circulation, massive production of estrin sets in rather acutely. The body rids itself of this excess by rapidly excreting it with the urine, as it is

not required in pregnancy; however, the amount steadily increases throughout pregnancy, so that in the first trimester of pregnancy one litre of urine contains about 2,000 to 3,000 mouse units; towards the middle of pregnancy, 10,000 units, and just before parturition, 50,000 units. Many patients with atrophic rhinitis have stated that their nasal condition improved for a time during their pregnancies, and, indeed, the advice of older practitioners, who told their patients with atrophic rhinitis to get married, may not have been amiss; however, the massive secretion of estrin does not depend upon pregnancy, since this hormonal increase occurs only in man, monkeys and the equidae, but not in other mammals. The greatest amounts of estrin are found in the urine of male animals; the urine of stallions, for example, contains over 170,000 mouse units per litre, and the female hormone is prepared by the testicles, since sexually immature stallions and geldings excrete but insignificant amounts. Paradoxical though it may appear, the testicle of the stallion contains over 500 times as much female hormone as the ovary of the sexually mature mare.

Estrin, likewise, occurs in other sites, being very extensively found in Nature. It is found in flowers and plants, bitumen, and unicellular organisms, as well as in young bacterial cultures, in which it acts as a general growth hormone.

In answer to the question, "What is the effect of introducing female hormone into the male?" Burrows reports that the continued administration of estrin to the male mouse causes an epithelial metaplasia in the duct and body of the bulbourethral (Cowper's) gland. Burrows also found that male mice after prolonged administration of estrin become resistant to that hormone, and that the resistance is accompanied by great development and activity of the interstitial tissue of the testis with widespread disappearance of the tubular epithelium. Rush, however, found that, although these long-continued administrations of the drug induced a metaplasia of the glandular epithelium of the prostate in mice, and caused an increase in the fibromuscular stroma of the gland, the administration of testosterone, the male hormone comparable to estrin, after these pathological changes had been established, induced a reversal of the metaplasia toward the normal type, but had little effect on the fibromuscular stroma.

These effects in experimental animals do not, in my opinion, contraindicate the use of estrin in the treatment of male patients with atrophic rhinitis, since a dosage of estrin comparable to that used in these experiments would be equal to about one-millionth of the body weight of the individual, administered daily for a period of about 70 years.

Recently also have come reports from Albany by Hamilton, who noted changes in the nasal mucosa of monkeys and eight humans after using testosterone propionate, the male hormone. The changes consisted of congestion, swelling and fluid formation grossly, and perivascular edema histologically. The use of these androgens, or male hormones, however, seems to be contraindicated in females since they exert a direct masculinizing effect, according to Hamilton; they inhibit menstruation, and there is a marked development of Skene's ducts into a prostatic type of organ. The chemical similarity of the female sex hormone and the carcinogenic hydrocarbons has given rise to much speculation and experimentation, particularly since the discovery that some of the carcinogenic hydrocarbons were estrogenic. Cramer, Perry and Ginzton, and Gardner, Smith, Strong and Allen have all reported the formation of sarcomas, carcinomas and benign epithelial proliferations in mice treated over long periods with estrin and its allied chemical substances. These malignant growths were not limited to any one particular organ, but have been reported in the breast, uterus, skin, alimentary tract and lungs. Cramer states that "unlike any other carcinogenic substance so far studied experimentally, the carcinogenic agent is not responsible, but a specific physiological sensitiveness to it. The action of estrin resembles that of other carcinogenic agents in reproducing, first, a hyperplasia of the tissues in which the cancer subsequently develops — the precancerous condition — and in the long period of time necessary to induce cancer."

Here again, we cannot exclude the possibility that we may be inducing cancer in these patients by treating their nasal mucosa with estrin, but it seems most unlikely that cancer will develop in these cases in the dosages that have thus far been used.

It is interesting to note that both the male and the female hormones apparently have a definite effect upon the nasal

mucosa. It has been mentioned that the stallion excretes a large amount of female hormone. The female hormone has, therefore, been regarded by Zondek as a decomposition product of the male hormone. "The presence of great quantities of estrin in the stallion's testicles indicates considerable intensity of production of the male hormone, accompanied by its correspondingly intensive decomposition to the female hormone. In every body both sex hormones are present. According to this theory, the female hormone present in the male body would be a decomposition product of the male hormone; conversely, the male hormone in the female body would be a preliminary stage in the natural synthesis of the female hormone." This concept is further supported by Butenandt's view that both hormones arise through progressive decomposition of the sterols.

A further study of this inter-relationship of the two hormones brings up the question as to whether the molecular structure of the hormones remains unchanged throughout the lifetime of the individual, and whether during periods of development corresponding to various ages, the hormone molecule might be chemically changed into higher or lower stages. If such changes occurred, the biological activity of the hormone would be altered, such processes leading occasionally to the phenomenon of disease. This supposition has been mentioned by other authors in connection with the problem of malignant diseases which are known to occur most commonly during or soon after the climacteric. Might it not be that such a change in the biological activity of the hormone contributes to the formation of nasal pathology and menstrual abnormalities encountered in patients with atrophic rhinitis?

In this series of cases I have not been able to find evidence of pituitary dysfunction in any of the cases by X-ray. Collip, Mortimer and Wright have reported a definite relationship between pituitary dysfunction and atrophic rhinitis; in my series of more than 70 cases, not one of the skulls has shown any evidence of pituitary abnormality. In 1927, Hoople and Rowe reported 80 cases of atrophic rhinitis in an attempt to find an endocrine factor, and concluded that there was no evidence obtained that endocrine glands had a causal relation to atrophic rhinitis.

Method of Treatment: The crusts are first removed from the patient's nose. This is done usually by removing them with bayonet forceps and then swabbing out the mucus that remains in the nose with pledgets of cotton. The treatment in the clinic at first consisted of painting the mucosa of the nose as thoroughly as possible with estrin, dipping a small cotton pledget into a 1 cc. ampoule containing 10,000 units of estrin. (The 2,000 unit size was first used, but better results were obtained with the larger size.) This method was quite efficacious, and seems well adapted to office practice where the number of patients to be treated is small; however, as the number of cases in the series increased, it was found that the use of pure estrin in the compressed air spray was a great time saver. For this spray, estrin, 10,000 units per cubic centimetre, is used and about 1 cc. sprayed in each nostril. The estrin for use in this manner was provided in the form of Amniotin in Oil gratis by E. R. Squibb and Sons.

For home use the patient is given the spray as follows:

Estrin, 10,000 U. 2.0

Sesame oil ad 30.0

Sig: Spray nose thoroughly t.i.d.

Since most of these patients have been accustomed to using nasal douches or irrigations for some time, and feel that they cannot get along without them, they are allowed to continue them for one or two weeks, and then these irrigations are gradually diminished in number and frequency. By the end of eight weeks the patient is usually irrigating the nose only once a week, and as soon as feasible even this is discontinued.

At about the end of six or eight weeks the amount of estrin used in the spray at home is cut in half, i.e., 10,000 units of estrin per ounce are given instead of the 20,000 previously used. Up to this time the patients have been seen at weekly intervals, and given the regular course of clinic treatment, consisting of the spray with 10,000 units per cubic centimetre, or of local applications of the same strength. As the patient progresses, usually after eight weeks, the clinic visits are gradually lengthened to two weeks, then three, four and six-week intervals. At each of these visits the estrin is used locally. At the end of three months many of the patients are able to get along without crusts or odor, and without nasal

irrigations, by using the spray at home only once or twice a day.

Exacerbations do occur in which there is increased crusting and odor. In women, these usually occur just before and during the regular menstrual periods, but increasing the dosage (the frequency) of the spray at home tends to alleviate these episodes somewhat. Other remissions seen less frequently in both male and female patients are attacks of mild sinusitis, at which time a purulent discharge may be seen, as well as the crusts. Unless the symptoms of the intercurrent infection become marked the patient continues his regime as outlined.

Contraindications and Side-reactions: At the present time the only contraindication is the onset of a severe acute sinusitis, which has occurred in a few of the cases, and which is apparently somewhat aggravated by the estrin. Even pregnancy, should this occur, would not, in my opinion, be adversely influenced by this form of therapy, in the amounts used. In this series there have been no cases in which pregnancy occurred during the treatment.

The worst side-reaction that occurs is headache. It is of a severe type, that does not yield readily to salicylates, and occurs generally at night, occasionally even waking the patient from sleep. These headaches have occurred in a small number of the cases, and with about equal frequency in males and females. The headaches have usually occurred on the night following the visit to the clinic, and are probably due to over-dosage of estrin.

A few patients have complained of nausea, usually following the use of the spray. They objected to the "tastelessness" of the spray when it dropped into the throat. The addition of aromatic oils, as oil of cinnamon and oil of clove, in small amounts served to overcome this undesirable feature.

More recently, reports have come of hypersensitivity to sesame oil. Certain patients, therefore, may be found who will react unfavorably when estrin is used with sesame oil as the vehicle. There have been no such allergic manifestations in the cases in this series.

It has been observed that all cases of atrophic rhinitis are not the same. Some patients have the crusts and the fetid

odor which has been described in the literature and which is only too familiar to all rhinologists, while other patients having an equal amount of crusting have little or no odor, and when this odor is present it is disagreeable, but not as nauseating as the former. Furthermore, patients who fall into this latter group seem invariably to have acquired the condition later in life, or at least after puberty.

For the purposes of this paper I have divided the cases into two groups. In Group I are those patients who noted the onset of symptoms before the age of 20 years, and in Group II those who at first had symptoms after 20 years of age. Those in Group I may be called true atrophic rhinitis, while those in Group II may be called secondary atrophic rhinitis. By setting 20 years of age arbitrarily as the line between the two varieties, there is probably considerable overlapping, and a few cases may reasonably occur in one group which should be in the other.

In Group I there are 25 females and 17 males; and in Group II, 12 females and four males. Actually, there have been about 75 patients who have received treatment, but only 60 are considered here. The remaining 15 patients have not been included for one of two reasons: some of these patients have a long history and treatment for lues; others have not been faithful, either in their home treatment or their visits to the clinic, and so could not be considered on an equal basis with the other patients.

It is my belief that the patients in Group II, who are classified as secondary atrophic rhinitis, have their nasal symptoms as a result of either lues or sinusitis primarily. Other factors, as anatomical abnormalities of the septum, etc., may also play a part.

In the accompanying charts it will be seen that all but six patients have shown an improvement. This data is the result of the impressions of one observer, and, consequently, another observer would probably occasionally disagree.

Results of Estrin Treatment: Of the 44 cases in Group I, all but six patients have been improved; in other words, 86.4 per cent of the cases were improved, and 13.6 per cent showed no improvement whatever. A great many of these patients have been treated by other methods in this and other hos-

pitals, and the unanimous opinion of these patients is that estrin therapy has helped them most.

Many of the cases marked as improved slightly (1+) still show some crusting, particularly in the ethmoid areas, but very few of them have any odor. A large number of these patients had such a marked degree of crusting that entire casts of the nose could be removed. Not one of the cases marked with the plus sign has as marked crusting at present as at the beginning of treatment. Four of the cases, marked with three plus signs, have shown a very marked improvement. In deciding whether or not these patients showed definite clinical improvement, each patient is asked exactly how many irrigations were used in the preceding week, and whether or not the nose was irrigated on the day of the clinic visit. Patients who previously used three nasal irrigations a day and who are now irrigating twice a week, and remaining relatively free from crusts, are tabulated as improved; but patients irrigating the nose daily before treatment and still continuing to do so have been marked with a question mark and have been considered individually. These patients really fall into the class of unimproved cases, even though the nose may be entirely clean on the clinic visit.

A few of the cases in the series seem worthy of individual mention. Patient No. 13 in Group I has been marked as questionably improved. When first seen this patient presented one of the worst cases of ozena I have ever seen. She is quite feeble-minded, and several years ago an attempt was made to place her in an institution. She has a large perforation of the nasal septum, a marked squint, terrible oral hygiene and, in general, is in very poor health. At present she is still irrigating the nose daily, and has very little crusting and no odor. Inasmuch as she has been irrigating the nose more or less for years, I cannot think that these irrigations alone are responsible for the diminution in the ozena which makes the patient tolerable at present; however, the treatment of such a case seems almost hopeless.

A second case which is marked as showing questionable improvement is patient No. 31 in Group I. Before this patient began treatment his ozena was so bad that he had not been able to eat at the same table with his family for four years. He is a boy of rather subnormal intelligence, who works

nights in the dusty atmosphere of a carpet factory. He still is obliged to irrigate his nose daily, perhaps because of his occupation, but his crusting and odor have diminished to such an extent that for the past five months he has no longer been objectionable to his parents.

The six cases that have not improved should be briefly mentioned. Patient No. 9 has a bilateral pansinusitis, and was operated for that condition about three months ago. All sinuses are still filled with pus by X-ray, and there has been no improvement in the amount of crusting or odor.

Patient No. 16 had no crusts or odor when first seen, and has none at present. The marked atrophy which was noted on the first visit has remained unchanged.

Patient No. 27 has not followed her treatment at home faithfully and is irregular in her clinic visits. On some occasions the nose has looked clean and at other times there were many crusts present. On the whole, there has been little if any improvement.

Patient No. 32 is a man, age 70 years, who has had atrophic rhinitis for over 50 years. This patient presented a moderate degree of crusting and odor and marked atrophy on his first visit. It seemed unusual that crusting should still be present after 50 years. According to the patient, he irrigates his nose each Sunday before going to church, to be sure not "to offend anyone." He has no crusts or odor at present, and yet there has been no visible change in the appearance of the nasal mucosa. The patient states that his nose feels more moist with the estrin spray, but it is my opinion that the same results would have been obtained with any oily spray.

Patient No. 35 is a boy who has not improved in four months of treatment. He is very irregular in his visits to the clinic, and probably his home therapy is just as irregular.

Patient No. 38, like patient No. 9, has pus in all sinuses, including the sphenoid, by X-ray. This patient has been advised to have an operation on his sinuses, because his nose is so constantly filled with pus and crusts that the spray is just wasted.

It was stated above that many patients seen with atrophic rhinitis were found to have syphilis, and, although some of

them were treated, they were not included in this series; however, two of the cases in this series show a positive Hinton test.

Patient No. 15 has had atrophic rhinitis for about 10 years. He contracted his luetic infection about two years ago, and for the past year and a half has been under treatment in the skin clinic. Patient No. 37, on the other hand, has had his atrophic rhinitis for about 25 years. Eighteen months ago this patient was brought to a hospital with a bleeding gastric ulcer, and an emergency blood transfusion was given, using the patient's brother-in-law as the donor. No Hinton or Wassermann was done on the donor, who was found later to be luetic, and the patient's blood was subsequently found to be positive, where it was definitely known to have been negative before the transfusion. This patient has been receiving vigorous antiluetic therapy since that time. Because the atrophic rhinitis in both these cases definitely antedated the lues by a considerable length of time they have been included in this series.

The patients in Group II, on the whole, seem to have done a little better than those in Group I. In these cases, chronic sinus infection and abnormalities of the nose seem to play a part. All of the cases in this group have shown some improvement. It is my opinion that correction of existing sinus conditions in the simplest way possible, and correction of other abnormalities, such as a deviated septum, will further improve this group of patients.

A number of the patients in the series have had biopsies taken from either the middle or inferior turbinates. It is regrettable that none of the photomicrographs enclosed with this paper show the nasal mucosa before treatment. Biopsies were not taken until the latter part of November and early December, and there seems to be little change in the mucosa before at least six months of treatment; however, at the present time biopsies are being taken on all new cases before treatment is started, so that in a few months from now, after a sufficient period of treatment has elapsed, second biopsies will be taken, which can be compared with those before treatment.

It is impossible, therefore, at present to state whether there is a definite change in the character of the mucosa as a result

of this type of therapy. Clinically, the mucosa seems to lose its blanched, grayish-white appearance and becomes pinker and more nearly normal in color, but the large airways have not diminished in size. Occasionally attacks of epistaxis occur. After several months of treatment there is a thin mucus discharge seen throughout the nose. Microscopically, there seems to be an increased vascularity of the tissues, and perhaps an increase in the glandular elements. The fibrous tissue which always characterizes the mucosa of atrophic rhinitis is unchanged, and undoubtedly will always remain so. As far as the epithelium itself is concerned, most cases show a stratified squamous type of epithelium, which in a few cases seems to be piling up in islands. Just what the end-result will be it is impossible at this time to predict.

Clinically, these patients are improved, and in a few cases the improvement has been nothing short of spectacular. In my experience I have never encountered a more grateful and appreciative group of patients; however, I do not believe that estrin therapy is a "cure" for atrophic rhinitis, in the sense that we cure other diseases. It may be that we can control the disease better with estrin than with other forms of treatment, just as insulin controls diabetes but does not cure it. In any event, patients who have had the disease only a few years respond better than those who have had their pathology established for many years, while, in general, it may be said that females react more quickly to estrin than do the males.

SUMMARY.

A brief review of some of the newer facts concerning the action and nature of estrin is given. Mention is made of its carcinogenic properties, as well as the effects produced experimentally in male animals. In spite of these last two facts, and because of the inter-relationship of the male and female hormones, it is believed that no contraindication exists for the use of estrin as a nasal spray. The male hormone used intranasally has produced a local hyperemia and perivascular edema in man and monkeys, according to Hamilton. Because of the physiological similarity in nasal and vaginal mucosa, and because of the pathological similarity in atrophic rhinitis and atrophic vaginitis, estrin has been used in the nose.

GROUP I.

Case No.	Name	Sex	Age	Age at Onset	Began Eserin Treatment	Abnormalities of Nose	Degree of Crusting and Odor on First Visit	Degree of Atrophy on First Visit	Degree of Crusting and Odor at Present	Nasal Irrigations at Present	Degree of Improvement	Addenda
1.	A.C.	F.	26	16	1-4-38	L. frontal chronic	++	++	Pus	0	+	
2.	C.A.	F.	29	9	1-15-38		++	+	+	0	+	
3.	E.B.	F.	23	18	9-13-27		+	+	0	0	+	
4.	G.B.	M.	5	2	11-1-37		++	+	+	0	+	
5.	M.A.	F.	21	9	12-11-37		++	++	0	0	+	
6.	G.C.	M.	24	9	12-9-37	Marked dev. septum	++	++	+	0	+	
7.	C.B.	F.	30	14	11-18-37		++	++	0	0	+	
8.	F.F.	F.	18	16	11-1-37	Dev. septum	++	+	+	0	+	
9.	E.B.	F.	25	17	1-8-38	Pansinusitis, bilateral	++	++	+	2 x week	0	Operated
10.	W.C.	M.	21	17	10-16-37	Chr. sinusitis, bilateral	++	++	0	0	+	
11.	L.G.	F.	31	8	9-28-37		++	++	0	2 x week	+	
12.	C.C.	F.	20	10	10-16-37		++	++	0	0	+	
13.	J.B.	F.	28	18	9-24-37	Septal perforation	++	++	+	Daily	+	
14.	A.B.	F.	36	13	9-9-37	Chr. thickening of sinuses	++	++	0	1 x week	+	
15.	H.G.	M.	25	15	10-1-37	Marked dev. septum	+	+	0	0	+	Lues
16.	C.C.	F.	43	18	10-3-37		0	++	0	0	0	
17.	M.D.	F.	39	14	11-18-37	Chr. thickening of sinuses	+	+	0	0	+	
18.	C.D.	F.	19	11	9-15-37	Chr. thickening of sinuses	++	++	+	0	+	
19.	A.P.	M.	21	12	1-22-38		++	++	+	0	+	
20.	M.G.	F.	16	12	8-16-37		++	++	+	0	+	
21.	F.M.	M.	20	16	1-28-38		++	++	+	0	+	
22.	C.C.	F.	25	15	10-16-37	Dev. septum	0	++	0	0	+	
23.	M.A.	M.	26	16	9-25-37	Dev. sept., chr. sinusitis	++	++	0	0	+	

GROUP I (Continued).

Case No.	Name	Sex	Age Onset	Age at Treatment	Began Estrin	Abnormalities of Nose	Degree of Crusting and Odor on First Visit	Degree of Atrophy on First Visit	Degree of Crusting and Odor at Present	Nasal Irritations at Present	Degree of Improvement	Addenda
24.	A.R.	M.	25	17	10-16-37	Dev. septum	++	++	0	0	+++	
25.	M.S.	F.	14	11	10-21-37		+++	+	0	0	+++	
26.	N.F.	M.	22	19	8-21-37	Dev. septum	+++	+	0	0	+++	
27.	A.R.	F.	29	19	12-30-37		+++	+	+	0	0	
28.	G.M.	M.	41	14	12-14-37		+	+++	0	0	+++	
29.	L.M.	M.	33	15	11-13-37	Chr. thickening of sinuses ³	+	+++	0	0	+	Visits irregular
30.	W.M.	M.	29	17	1-29-38		+	+++	0	0	+	
31.	A.M.	M.	20	14	8-30-37		+++	+++	+	Daily	?	
32.	G.P.	M.	70	14	10-14-37		++	+++	0	1 x week	0	
33.	G.J.	F.	10	7	11-22-37		+++	+++	+	0	++	
34.	G.M.	F.	49	19	10-28-37		+	+++	0	0	+	
35.	V.G.	M.	16	12	12-30-37		+	+++	+	0	0	
36.	A.S.	F.	15	13	12-30-37		+	+	0	0	+	
37.	C.S.	M.	42	17	10-28-37		+	+++	+	0	+	Lues
38.	W.S.	M.	35	10	1-11-38	Chr. sinusitis, bilateral	+++	+++	++	0	0	
39.	A.K.	M.	37	12	1-11-38	Dev. sept., chr. thk. of sinuses	++	+++	+	0	+	
40.	T.B.	F.	30	16	10-9-37		++	++	0	0	++	
41.	J.Z.	M.	10	8	11-20-37		++	+	0	0	++	
42.	M.W.	F.	21	17	12-18-37	Chr. thickening of sinuses	+++	+	0	0	++	
43.	J.T.	F.	18	15	1-28-38		+++	++	+	0	+	
44.	M.P.	F.	43	18	1-21-38	Chr. thickening of sinuses	+++	+	0	0	++	

Total, Group I—35 females, 19 males.

GROUP II.

Case No.	Name	Sex	Age	Age at Onset	Began Estrin Treatment	Abnormalities of Nose	Degree of Crusting and Odor on First Visit	Degree of Atrophy on First Visit	Degree of Crusting at Present	Nasal Irritations at Present	Degree of Impediment	Addenda
1.	M.D.	F.	33	23	11-16-37		++	+	0	1 x week	++	
2.	C.C.	M.	65	35	10- 8-37		++	+	0	0	++	
3.	O.B.	F.	58	47	9- 9-37	Dev. septum	++	+	+	0?	++	Ethmoidec., 1927
4.	L.S.	F.	41	37	10- 5-37	Dev. septum	+	++	0	0	++	Visits Irregular
5.	A.G.	M.	43	38	11-19-37	Dev. septum	+	+	0	0	++	
6.	R.K.	F.	43	26	1- 4-38		++	+	0	0	++	
7.	E.L.	F.	54	44	11-16-37		++	+	+	0	++	
8.	M.L.	F.	60	49	10- 4-37	Dev. septum	++	++	+	0	++	
9.	A.L.	F.	50	35	9-21-37	Pansinusitis, bilateral	++	++	0	0	++	
10.	E.G.	F.	32	22	10- 8-37		++	++	0	0	++	
11.	M.P.	F.	50	44	9- 8-37		++	+	0	0	++	
12.	E.O.	F.	45	30	2- 7-37	Dev. septum	++	+	0	2 x week	++	
13.	M.P.	M.	35	32	9-30-37		++	+	0	0	++	
14.	G.D.	M.	66	36	11-15-37	Dev. sept. with perforation	++	+	0	2 x week	++	
15.	B.G.	F.	45	42	9-29-37		++	+	+	0	++	
16.	M.W.	F.	44	29	11- 1-37		++	+	0	0	++	

Total, Group II—12 females,
4 males.

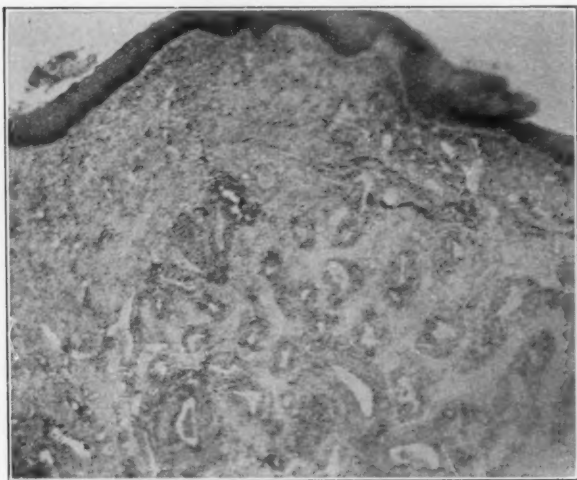


Fig. 1. Low power section through right inferior turbinate showing moderate number of blood spaces with thick walls, glands absent, and stratified squamous epithelium. Considerable connective tissue fibrosis. Patient No. 12; six months' treatment.

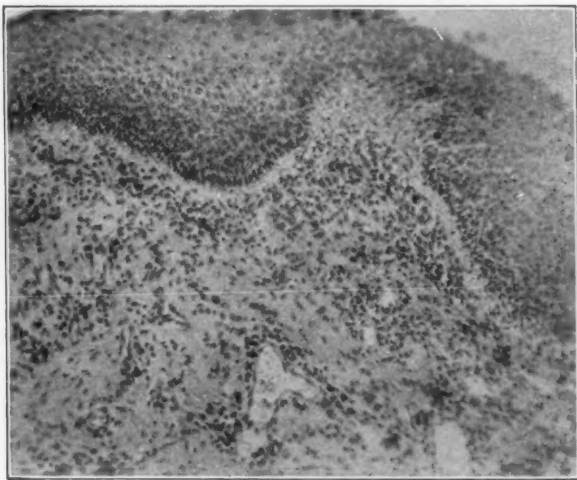


Fig. 2. High power of the same section.

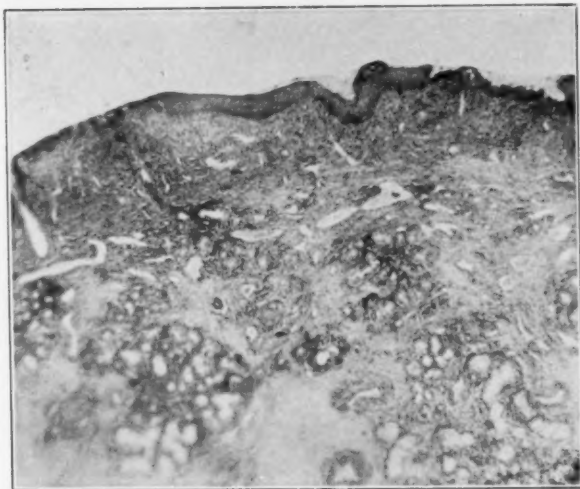


Fig. 3. Section (low power) from the left inferior turbinate of Case 26, showing stratified squamous epithellum, with small islands of more or less columnar cells, numerous glands and blood vessels. There is considerable thickening and infiltration under the epithellum. After six months' treatment.

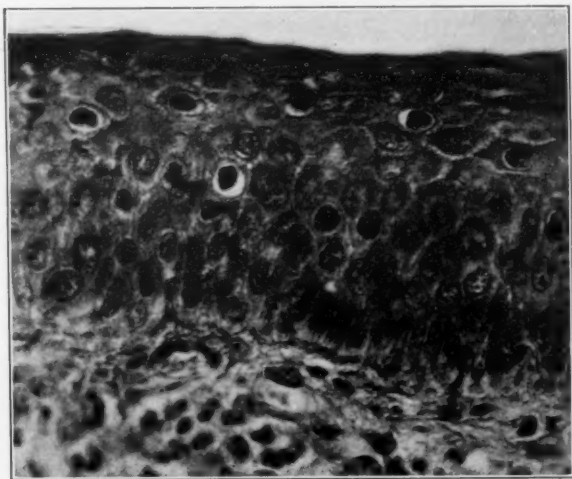


Fig. 4. High power section of the same, taken through one of the "islands."

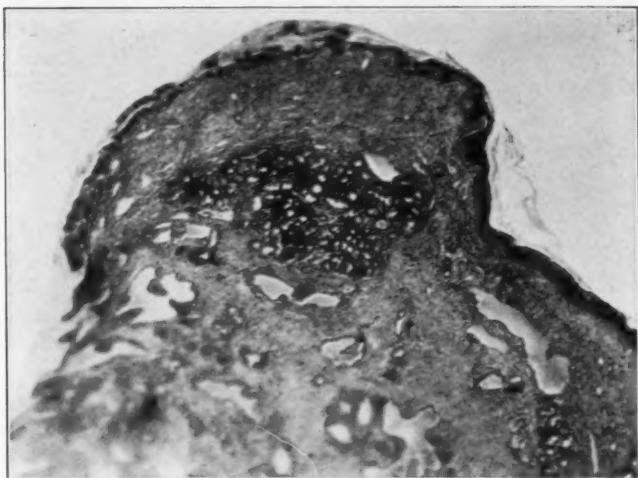


Fig. 5. Low power section of right inferior turbinate of Case 10 (Gr. II) after six months' treatment, showing thick blood vessels, thin stratified squamous epithellum, thickened basement membrane, and thick walled blood vessels. Chronic inflammation.



Fig. 6. High power of the same section.

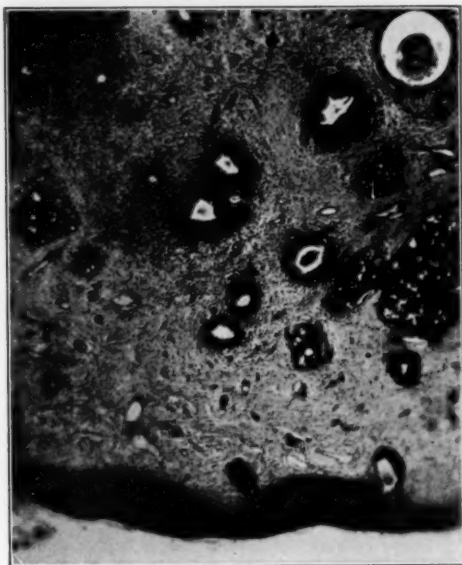


Fig. 7. Low power section. Case 40. Stratified squamous epithelium, thick walled blood vessels, and considerable fibrosis. Three months' treatment.

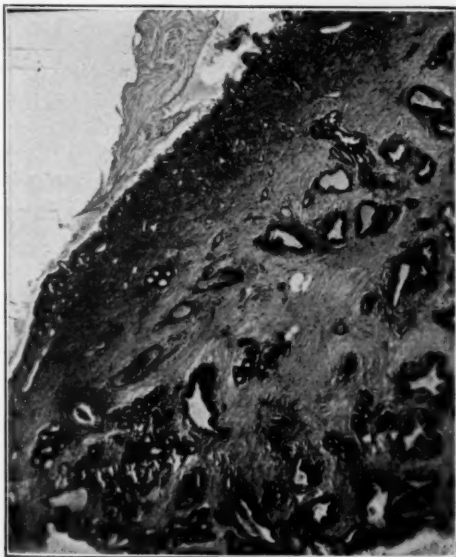


Fig. 8. Case 15. Low power. Very thin epithelium, numerous blood vessels and glands, and moderate amount of fibrosis.

A series of 60 cases is presented, divided into two groups; the first, consisting of patients with an onset before 20 years, is termed true atrophic rhinitis, and the second group, made up of patients with onset of symptoms after 20 years of age, is called secondary atrophic rhinitis.

There was clinical improvement as shown by diminution of crusts and odor in 86.4 per cent of the cases in Group I, and all patients in Group II were improved.

A few photomicrographs of biopsies from these patients are presented, but all biopsies are after several months of treatment and none were taken prior to treatment.

No mention is made of menstrual abnormalities in these patients, although these are known to occur frequently, and, also, a few have improved during the treatment. A competent gynecologist is necessary to give these symptoms their true value.

Likewise, no mention has been made of the incidence of heredity in the disease, nor the incidence of previous intranasal surgery in these cases, both of which are probably factors in its causation.

Because of its wide variety of effects upon the body, aside from its function as a sex hormone, it can readily be seen that estrin, properly used, is one of the greatest weapons which we have for combating diseases and physiological conditions which heretofore have been obscure. Besides acting as a hormone, estrin acts also like a drug, and its action is more far-reaching than insulin, more powerful than adrenalin, and more spectacular than ergot. As the study of this substance progresses, its exact place in the armamentarium of the otolaryngologist will undoubtedly be found.

CONCLUSIONS.

1. Estrin therapy in atrophic rhinitis has given clinical improvement of symptoms.
2. Side-reactions and exacerbations do occur, but, in general, patients remain symptom-free as long as estrin is used.
3. Biopsies should be taken before and after treatment, in order to determine the extent of the changes, if any, in the nasal mucosa.

4. Determinations of estrin in the urine should be carried out before treatment, and periodically thereafter.

5. The relationship between menstrual abnormalities and atrophic rhinitis should be undertaken by a gynecologist.

6. The exact mechanism by which estrin acts upon the nose is not known, and the extent of changes in the mucosa, as well as the permanency of the improvement clinically, can be determined only after years of careful study of these cases.

243 Charles Street.

**CONCUSSION SOUND WAVES FROM LARGE GUNS
IN ACTION. AUTHOR'S ABSTRACT.***

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In April, 1918, Col. R. A. Millikan, Chief of the Department of Science and Research of the Council of National Defense (the National Research Council), requested the writer to undertake the study of the physical characteristics of the sound waves produced in the atmosphere near large guns in action. Quantitative information in regard to these effects was desired by Dr. D. R. Hooker, of the Department of Physiology of Johns Hopkins University, who, acting under the same auspices, was engaged in an investigation of the physiological effects of air concussion, which, at the time of the World War, was popularly supposed to be the cause of the malady vaguely referred to as "shell shock." Case School of Applied Science granted a leave of absence from teaching duties from April, 1918, to November, 1919, and experiments were carried out, at first in the laboratory at Cleveland and later at Sandy Hook Proving Ground under permission and authority of Gen. C. C. Williams, Chief of Ordnance. The commanding officers of the Proving Ground were very sympathetic with these scientific experiments, and permitted a most liberal use of the facilities of the Proving Ground, with only the restriction that the regular work of the Proving Ground should not be interefered with. Col. Millikan assigned several enlisted scientific men as assistants, and at the Proving Ground any required help was supplied. Private laboratory rooms were furnished, and the machine shops, transportation and construction services were made available. The Chief Proof Officer gave advance notice of the programs of firing of large guns, and at times arranged the programs to accommodate the placing of recording apparatus and the making of observations. All conditions combined to provide a most extraordinary outfit, as regards completeness, for the study

*Abstract of paper presented at the Seventy-first Annual Meeting of the American Otological Society, Inc., Atlantic City, May 5, 1933.

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of sound effects of various kinds. The conditions could hardly be reproduced, at any cost, upon orders.

Taking advantage of this remarkable situation, the researches were extended beyond the first purpose, and led to four distinct groups of results. These relate to: 1. the pressure effects in the air around large guns in action; 2. the form and physical characteristics of the sound waves from large guns; 3. propagation of the sound wave from the muzzle of a large gun; 4. the normal velocity of sound in free air. The reduction of the large mass of observational material relating to the sounds from large guns has only recently been completed.

It was necessary to devise several new instruments and devices for these unusual studies. The firing of several thousand "rounds" from the large guns was observed during the development of the methods, and for testing and calibrating the recording instruments. Quantitative records from 196 firings have been used in the definite results for pressures and velocities of sounds.

The writer had developed the Phonodeik for photographically recording ordinary sounds, such as those from musical instruments, and the voice. It was entirely practicable to adapt the Phonodeik for recording sound waves from large guns at stations where it was safe, from a physiological point of view, for a human being to work. The study of shell shock required that measurements be made at stations which were physiologically unsafe. The pressure produced in the explosive sound wave may vary from a few ounces to 1,000 pounds per square inch. No instrument for measuring such pressures was available, and experiments were undertaken, at first in the laboratory and later in the field, to develop an apparatus which would be simple but certain in action, portable and of such rugged construction as to be usable under the conditions of field artillery operation. The pressure gauge developed for this purpose may be called a baroscope. It is a kind of metal microphone apparatus in which the displacement of a diaphragm, due to a pressure, can be measured by means of a micrometer screw. A dozen such baroscopes were constructed in the shops, and these could be distributed over the field around a gun or around an animal being used for tests, in any direction or manner, and thus one could obtain 12 meas-

urements of the pressure of a single shot. It was demonstrated that the air concussion, due to the discharge of a 14-inch rifle, might produce a pressure on the ground in front of the gun as great as 500 pounds per square inch. Such a pressure would undoubtedly be fatal to a human being. It was shown that at distances greater than 50 feet from the gun the pressures are about five pounds per square inch, and are in no way dangerous. The measurements indicated that near the breech of the gun, where the gunners are ordinarily stationed, the pressures are rarely as great as two pounds per square inch.

An extended report of "The Physiological Effects of Air Concussion" has been made by Dr. D. R. Hooker in *The American Journal of Physiology*, Vol. 67, pp. 219-274, 1924. The following comments have been selected from Dr. Hooker's report:

"The data regarding the effects of gun blast on anesthetized dogs are insufficient to warrant a positive statement as to the concussion pressure requisite to produce shock. From the figures one may judge, however, that approximately 275 pounds per square inch (18 to 19 atmospheres) are necessary to establish primary shock in the case of 10-inch and 12-inch rifles. It is interesting to note that the 12-inch mortar, even when a pressure of 388 pounds per square inch was delivered, did not produce shock. Miller, in repeated observations, found that this gun, which is mounted somewhat nearer the ground than the rifles, gave uniformly high pressures. The only explanation which seems to accord with Miller's observations and my own results is that the duration of the high pressure phase in the case of the mortar is insufficient to overcome the physical resistance of the tissues. This hypothesis is supported by the fact that 288 pounds before the 10-inch rifle was less effective than 267 pounds before the 12-inch rifle, the latter presumably having the longer phase of high pressure. . . Exposure of animals, 10 to 20 feet in front of 10-inch and 12-inch rifles, yielding a concussion pressure of 18 to 19 atmospheres, usually produced primary shock. This condition is essentially instantaneous in onset. . . The concussion pressure adequate to produce shock results in extensive laceration of the tympanic membrane of the ears. . . There is no evidence that the functional capacity of the heart was affected, and the valves were uninjured. . . No evidence of hemorrhage, gross or petechial,

has been found in the nervous tissues. . . The blood is not hemolyzed. . . The lungs showed areas of red hepatization, especially in the lower lobes, which were practically solidified. . . It is open to question whether the condition of shock in these experiments is not primarily associated with brain concussion. . . The effects are due to the duration of the phase of high pressure rather than to the height of the atmospheric pressure as such, or to the tonal vibration of the atmosphere."

G. R. Marage, in France, made a study of the shocks of war, the results being presented at the Paris Academy of Sciences, *Comptes Rendus*, Vol. 166, pp. 132-135, 1918. Marage reports pressures close to the centre of explosion of the order of magnitude of 150 to 300 kg. per square centimetre. These pressures diminish rapidly and at distances of 30 metres are only 2 to 3 kg. per square centimetre. Marage reports the following clinical facts relating to the shock effects on human beings. Some die without receiving visible wounds. Others lose consciousness, speech, memory, sight or hearing, accompanied by intense headaches. These symptoms may continue for a few weeks or for years. The following explanation is made: The body acts like an elastic sack full of liquid, which communicates directly with an undeformable sphere, the skull, filled with an isostatic liquid in which floats the brain. An increase of exterior pressure is transmitted as a hydrostatic pressure to the interior of the skull and to the brain unless the liquid finds natural obstacles to such transmission, and unless the duration of the high pressure has been very brief; less than a hundredth of a second. Devices introduced into the external ear may protect the eardrum, but they will be absolutely ineffective against shocks.

The address was accompanied by illustrative diagrams, photographs of apparatus and of sound waves. Many of these illustrations, as well as more detailed accounts of the methods and instruments here briefly mentioned, have been given in a book by the author, "Sound Waves: Their Shape and Speed," recently published by the Macmillan Co., of New York.

Case School of Applied Science.

DISEASES OF THE MAXILLARY SINUS AND THEIR RELATIONSHIP TO THE ORAL CAVITY.*

DR. JOHN M. LORÉ, New York.

In the ordinary everyday practice of our specialty, we from time to time meet with intriguing problems of diagnosis and management. And not infrequently the problems involve the relationship of the maxillary sinus to the oral cavity, such as sinus tracts between the antrum of Highmore and the mouth, cysts, growths and fractures, not to mention various infections.

And it is for the consideration of these latter problems that this brief presentation is made.

The surgical anatomy is fairly well understood by the rhinologist; however, to freshen his memory, a few anatomical drawings taken from Skillern's "Accessory Sinuses of the Nose" will be shown.

Let us turn to the diseases of the maxillary sinus which bear some relationship to the oral cavity. It must be borne in mind from the outset that in some of these diseases the relationship is only one of subjective symptoms, rather than pathology. For example, one may have a "toothache" due entirely to an acute antrum, and another may have pain in the antral region or canine fossa due entirely to a diseased tooth.

Suppurative Diseases of the Maxillary Sinus: In the ordinary acute antrum, the only oral relationship may be pain in the teeth, as stated above. These cases give a history of a head cold, as characterized by a stuffy nose, sneezing and possibly headache, soon to be followed by a watery discharge, later becoming purulent, and then a localization of pain either in the antral region alone or referred to the teeth on the affected side, and sometimes to the frontal region or back of the ear on the same side. The nature of the symptoms immediately directs your attention to the maxillary sinus and the teeth. Nasal examination and transillumination of the sinuses

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will establish a tentative diagnosis. It must be remembered, however, that the teeth may be an etiological factor in the antral disease. The average acute maxillary sinusitis, uncomplicated by dental caries or apical abscesses, will usually respond to conservative intranasal treatment. Not infrequently, however, it becomes necessary to resort to lavage of the antrum. The pus obtained in the uncomplicated cases is usually in large lumps and odorless. Should a foul granular type of pus be obtained, it then becomes imperative to study the teeth which are in relationship to the antrum, and this is best done by competent X-ray studies. In these cases, it not infrequently is found that some diseased condition of the teeth exists. It is this type of acute antrum, complicated by dental disease, that will not clear up until the dental condition is corrected.

Occasionally we see a neglected case of acute maxillary sinusitis in which a spontaneous rupture has taken place into the canine fossa, producing considerable local swelling and tenderness. Raising the upper lip and inspection of the canine fossa will reveal considerable fulness, infiltration and edema at this point. Fluctuation may be demonstrated. Constitutionally, the patient usually appears sick. If an incision is made into this swelling, pus will be obtained. In these cases it is of the utmost importance to determine the condition of the sinus and the teeth on the affected side, for an apical abscess or an infected cyst may produce a similar clinical picture. In other words, a careful work-up of the case is necessary and a snapshot diagnosis deplored, for it is in these hastily diagnosed conditions that we get our disastrous sequelae. A careful work-up should consist of a history, careful intranasal examination, dental examination and X-ray studies of both the sinuses and the teeth. Transillumination, while useful, is not to be substituted for competent X-ray work. The X-rays of sinuses by the average X-ray laboratory which we see are so poor that they are actually misleading and worse than useless. Fortified by this work-up, we are in a position to differentiate between maxillary sinus disease, dental infection and cysts, and depending on the diagnosis so arrived at will depend the management and treatment of a given case.

In chronic antral disease, the problem is not unlike that in the acute conditions. The subjective symptoms, of course,

are less severe. Nasal and postnasal discharge in varying amounts, dull pains in the antral region occasionally referred to the teeth or ear, and frequent "head colds" or exacerbations are the nasal symptoms. Here, again, a careful work-up may reveal both antral and dental disease. In such cases, treatment of the one may cause a cure of the other, but not frequently. It used to be thought that most antral suppurative diseases were due to teeth, but it is now felt that not more than 20 per cent are due to this cause.

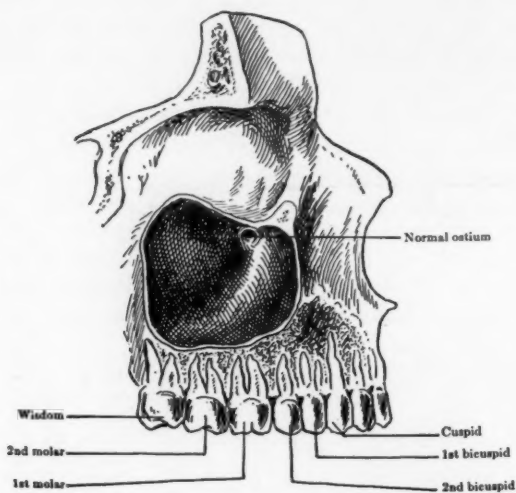


Fig. 1. Relation of the roots of the teeth to the floor of the maxillary sinus. Note position of normal ostium; also that the roof of the antrum is the floor of the orbit.

These chronic antra will not as a rule respond to nonsurgical intranasal treatments. When antral lavage, either by way of the natural orifice or by puncture, reveals a granular foul pus, it is best to at once consider intranasal antral surgery, consisting of a large opening into it through the inferior meatus. In the uncomplicated case, with no associated dental or ethmoidal disease, this simple operation with subsequent lavages will clear up this condition.

If in spite of adequate drainage, the discharge, and foul discharge in particular, persists, then direct your attention to the teeth. The entire explanation may be here. The removal of a diseased tooth may produce an immediate cure.

Let's reverse the condition. The patient visits the dentist and it is found that there is an apical abscess which requires extraction of the offending tooth. If the tooth does not communicate with the antrum, which in this case we assume to be diseased, the mere extraction will in all probabilities have little if any influence on the course of the antral suppuration. If, however, this tooth entered the antrum, its removal would produce a sinus tract communicating with the antrum. What should the subsequent care of these cases be? It must be



Fig. 2. Ostium divided by ridge of mucous membrane (Skillern's "Accessory Sinuses of the Nose").

admitted that in a few cases lavage of the antrum through the alveolar process will result in a cure of the antral disease and a subsequent closure of the sinus tract. If after a few washings little or no improvement is noted, then it becomes necessary to resort to other measures, because if these washings are continued, a chronic sinus tract will be the result.

My experience has been that if an intranasal antrotomy or so-called permanent opening is made into the antrum and the washings continued through this opening, the sinus tract will promptly close and the antrum clear up in most instances.

However, we occasionally meet with cases in which neither the antrum clears up nor the sinus tract closes up in spite of adequate drainage via the inferior meatus. What then? We either have a very large opening from the alveolar process into the antrum or a badly diseased condition of the mucous membrane in the antrum, or an osteomyelitis of the jaw or antrum, not to mention an associated ethmoiditis.

In cases of large sinus tracts associated with foul antra, plastic operations attempting to close the openings will fail. These are the cases which will require a radical antrum operation such as the Caldwell-Luc. This operation may cause the



FIG. 3. Thick, cancellated bone between the apex of the tooth root and the sinus floor.

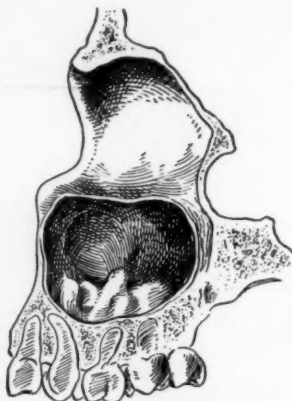


FIG. 4. Roots of teeth projecting into the maxillary sinus cavity.

(Skillern's "Accessory Sinuses of the Nose.")

sinus tract, if not too large, to close. But if it doesn't close, a plastic operation performed now will in all probabilities prove successful.

Of course, occasionally a closure is obtained by merely curetting the opening, after the antrum has been successfully treated. It won't work, however, where the tract is large. The Dunning operation for the closure of these openings has proven successful.

To cite a case: Mr. N. P. had a second molar removed because of a "toothache." The extraction was apparently well done, but the dentist found that the tooth communicated with the antrum. The antrum was washed several times through

the alveolar process, foul pus being obtained with each washing. When it was realized that the antral condition was not improving, he was referred for a sinus check-up. Nasal examination was essentially negative, no free pus being demonstrated in the nostril. He did give, however, a history of frequent colds with considerable postnasal drip. Transillumination showed the affected antrum to be black. A diagnostic lavage was performed by puncturing the inferior meatal wall, and a small amount of foul pus was obtained. An intranasal

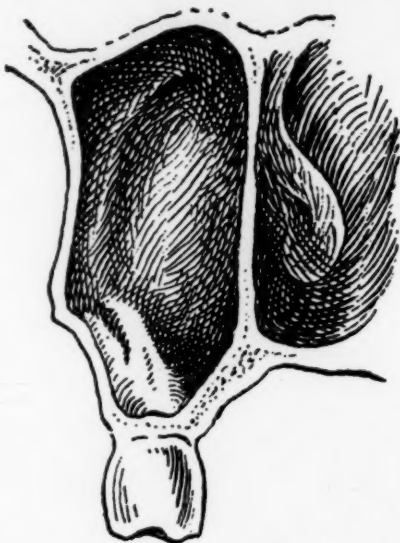


Fig. 5. Lateral view of tooth root projecting into the maxillary sinus (Skillern's "Accessory Sinuses of the Nose").

antrotomy was suggested and accepted by the patient. Contrary to our expectations, the operation did not clear up the antral pathology. X-rays taken now showed considerable clouding of the antrum, especially in the outer wall and floor. The sinus tract was still present. In view of this, a Caldwell-Luc operation was performed under local anesthesia. We soon realized why we had not obtained any result from the intranasal antrotomy. A dense fibrous mass was found in the antrum, extending from the sinus tract up to the roof of the antrum. When this was removed by curetting, it was found

to be hollow and the centre filled with foul pus. Pathological examination of the tissue removed showed it to be fibrous tissue. Within a few days, the sinus tract had closed and the second lavage was clear.

We occasionally see cases in which there is an opening above the alveolar margin, in the canine fossa, communicating with the antrum, and in which a history of radical surgery is usually obtained. The failure of an incision for the Caldwell-Luc operation to close is practically always due to a continuance of a diseased condition of the antrum or incomplete surgery. No amount of plastic closure will be successful until the antrum has been cleaned out and adequate drainage through the nose established.

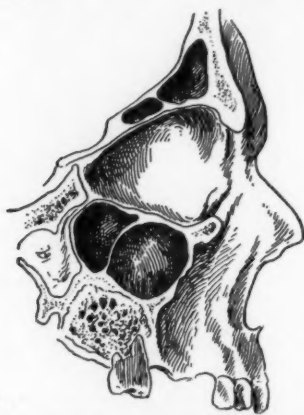


Fig. 6. Complete septum dividing antrum into an anterior and posterior compartment (Skillern's "Accessory Sinuses of the Nose").

Growths: The antrum is not infrequently the seat of a new growth, either benign or malignant. The benign ones usually met with are cysts and polypi. These bear no relation to the oral cavity except in the method of approach for their removal. In these cases the operation of choice is the Caldwell-Luc.

However, there are other benign growths not originating in the antrum which may involve it by encroaching on it, or which may be mistaken for antral growths. For example, the dentigerous cyst is occasionally not recognized by the rhinologist. This tumor is due to proliferation and degeneration of cells of the enamel organ before eruption of a tooth, the latter

lying partially or completely developed within the cyst cavity. The cavity is single and has a fibrous capsule lined with layers of squamous epithelium. It is filled with straw-colored fluid. The characteristic thing is that it contains the crown of an unerupted tooth. If located in the region of the antrum, it

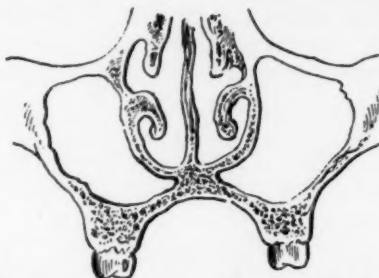


Fig. 7. Reabsorption of bone into the hard palate, bringing the maxillary sinuses into direct relation with the roof of the mouth. Note the relation of the floor of the maxillary sinus to the floor of the nose (Skillern's "Accessory Sinuses of the Nose").

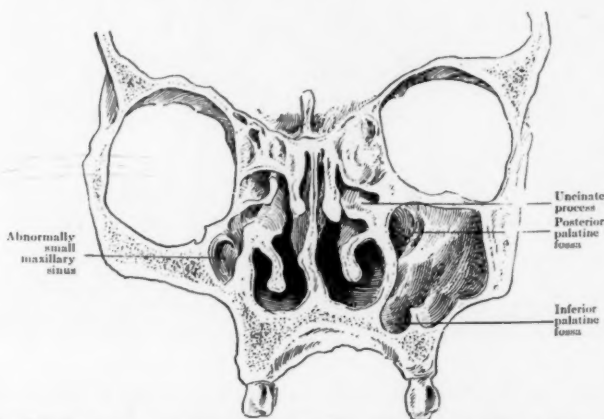


Fig. 8. Marked asymmetry of maxillary sinuses. Excessive enlargement on the left due to outward expansion of anterior and lateral walls. Right sinus under-developed (Skillern's "Accessory Sinuses of the Nose").

may so encroach on the floor of this sinus as to push it up, thus making the antral space very small. Rarely does such a cyst rupture spontaneously into the antrum. Diagnosis is made by determining the absence of a tooth and the X-ray findings, which will show a clear area due to the absence of bone, and having sharp, well defined edges. In addition, the

unerupted tooth will be seen in the cyst. If the growth has assumed a large size, it will have caused a thinning or absorption of the anterior wall, so much so as to fluctuate on palpation. The dentist is more familiar with this form of cyst and knows that complete removal with the capsule is the proper treatment. Unfortunately, the rhinologist will occasionally mistake it for an antral infection and will attempt to drain it by lavage or puncture. Occasionally these cysts undergo suppuration, thus complicating the clinical picture.

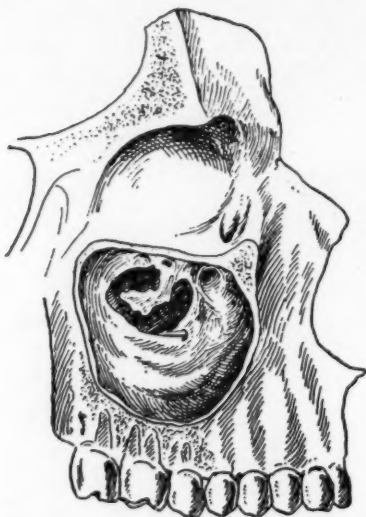


Fig. 9. View of maxillary sinus from without, showing place of exit of point of needle when introduced intranasally beneath the inferior turbinate at the usual position for exploratory needle puncture (Skillern's "Accessory Sinuses of the Nose").

Then, we have the dentoperiosteal cyst, which is commoner than any other form of cyst, in this region. In these cases the cause is entirely different from the dentigerous cyst. In almost all instances it is the result of a chronic inflammatory process about the apex of a tooth with dead pulp. In these cysts there are no unerupted teeth, and if a tooth is found to be missing it will be due to a previous extraction. If a dentoperiosteal cyst has already started, the removal of the offending tooth will not necessarily cure the cyst. This also has a fibrous capsule, lined with squamous epithelium. The

fluid in the cyst, however, contains cholesterol crystals. Like the dentigerous cyst, this may be large enough to encroach on the antral space, and may also undergo suppuration. If, after careful X-ray study and testing of teeth for vitality, the diagnosis is still in doubt, aspiration under aseptic conditions may be tried. This may differentiate it from a sarcoma.

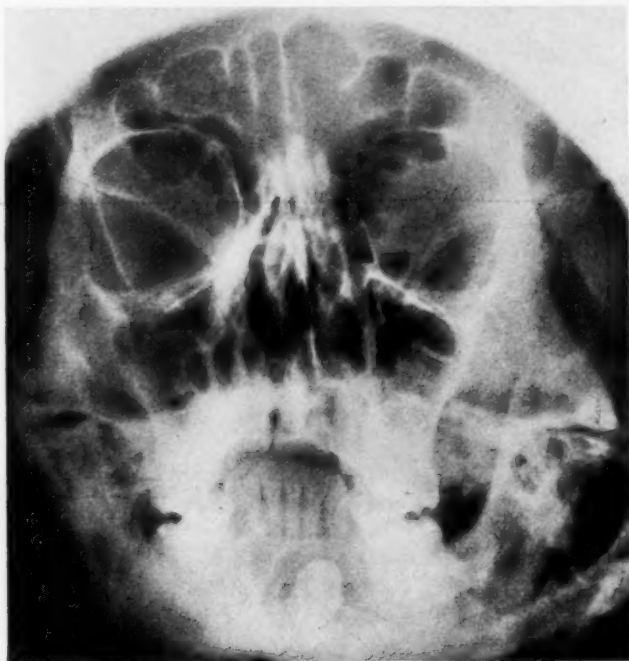


Fig. 10. Mucous cyst of maxillary sinus.

Here, again, incision and drainage for this type of cyst is mentioned only to be condemned. Removal of the offending tooth or teeth and removal of the cyst are the methods of choice.

Of the malignancies of the antrum, we must consider the sarcomata and carcinomata. Long before any external swelling is noted, there may be signs of trouble in the nose. Increasing nasal obstruction, unexplained bleeding and neu-

ralgic pains on the affected side require careful consideration. Intranasal examination may reveal a tumor mass in the region of the middle meatus or there may be a bulging of the nasal antral wall towards the nasal septum. X-rays of the sinuses may reveal beginning bone destruction. Later on, there may be fulness and swelling of the face in the canine region, with or without protrusion of the eye, and oral examination may



Fig. 11. Mucous cyst of maxillary sinus.

reveal a firm mass under the lip and filling the canine fossa. Definite diagnosis is established by biopsy.

The sarcomata lend themselves to radiation therapy. Surgery, in these cases, calls for resection of the superior maxilla and should be followed by radiation therapy.

In the cases of carcinoma of the antrum seen by me, there has always been some involvement of the ethmoids. These cases have been treated surgically by performing, first, a

Caldwell-Luc operation and then continuing with a transantral ethmoidectomy, followed by radium therapy. As a result of the radiation therapy, the Caldwell-Luc incision often fails to heal, which may be a blessing in disguise, and occasionally

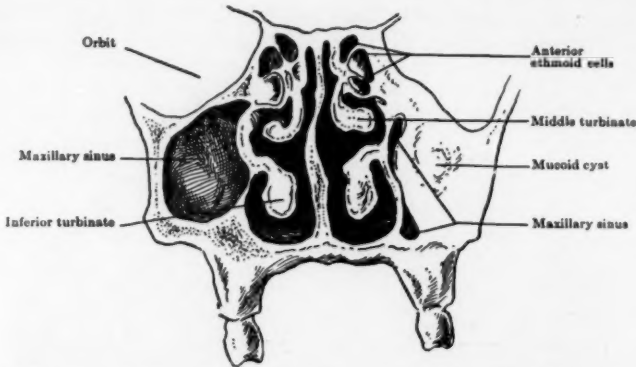


Fig. 12. Large glandular mucoid cyst almost filling antrum of left side (Skillern's "Accessory Sinuses of the Nose").

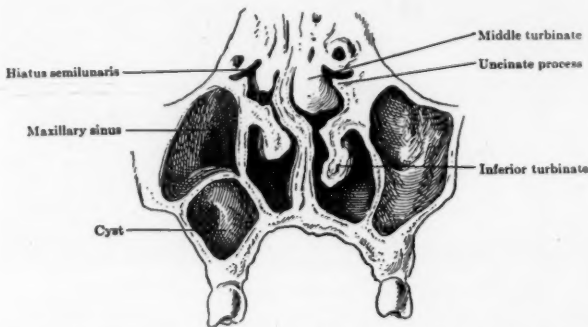


Fig. 13. Dentigerous or true bone cyst of an antrum on right side (Skillern's "Accessory Sinuses of the Nose").

there is necrosis of the hard palate, with an eventual large communication between the antrum and mouth. In such cases the defect in the hard palate is covered by means of a proper plate. The thing of note to me in these cases of carcinoma which have had radium is the severity of the pain which the patient experiences for many weeks after its application.

A few words about injuries involving the antrum are in order. Not infrequently, fractures of the anterior wall of the antrum, the superior maxilla, or both, are seen.

Fractures of the anterior antral wall are caused by direct trauma and are associated with considerable local tissue reaction. In addition to local swelling of the cheek, there may be a large hematoma in the canine fossa, causing a protrusion

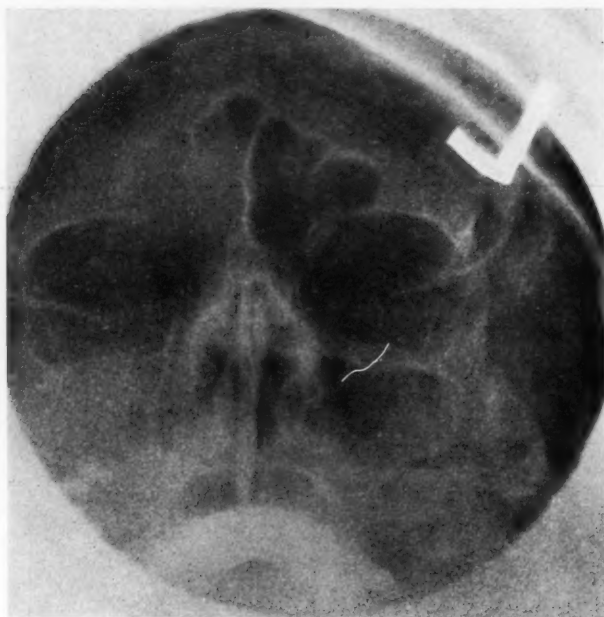


Fig. 14. Carcinoma of maxillary and ethmoid sinuses.

of the tissues forward and downward. Raising of the upper lip and inspection under it will reveal a fairly large discolored swelling, which fluctuates. In these cases we also find evidence of hemorrhage into the antrum. If the fracture has not involved the infraorbital rim, there will in all probability be no deformity and no reduction of the fractured anterior wall will be required. The treatment of the hematoma in the canine fossa and the hemorrhage into the antrum should be expectant. The thing to look for is infection. If none occurs, then leave

the hematoma and antrum alone. If it does occur, then the treatment resolves itself into one of draining the infected area. An incision as for the Caldwell-Luc operation will drain the canine fossa, and the antral lavage will drain the antrum.

In bilateral fractures of the superior maxilla in which the antrum or antra are involved, there will be a displacement downward of the upper jaw, with an accompanying distortion of the facial and nasal structures. Here prompt reduction and fixation are required. The Kingsley splint seems to be the one preferred and is best applied by the dentist. It is surprising, indeed, to note the change in the position of the facial and nasal structures when this type of fracture has been properly reduced.

Before concluding, a few words about the so-called intra-nasal antrotomy would not be amiss. Theoretically and on paper, the flap operations are pretty, but in actual practice they are tedious and somewhat lengthy, not to mention difficult. The procedure which we have been using is simple, short and effective. The initial opening into the antrum via the inferior meatus is made with the Faulkner bob, and, correctly used, makes a large opening. The opening is then enlarged by breaking down the inferior and anterior margins of this opening, finishing off by using the antral rasp on those same margins. No attempt is made to make a flap. In the subsequent treatment, antral sounds are used to keep the opening patent.

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325 West End Avenue.

**MODIFICATION OF THE SEMILUNAR GANGLION
APPROACH USED IN SURGERY OF
THE PETROUS PYRAMID.***

DR. MARVIN F. JONES, New York.

The problems created by petrouisitis are rapidly being solved. The exact location of a pathological process in the petrous pyramid is still difficult. Surgery has resolved itself into transpetrous procedures and methods which accomplish an entrance to the involved area through the walls. Most authorities agree upon the necessity of utilizing a method which seems best adaptable to the individual case. The operator is usually governed in his choice by the relative safety of procedures which will effectively drain the diseased area.

Some years ago I demonstrated an approach to the petrous pyramid, which was based on measurements using the eminentia arcuata as a landmark. Unfortunately, for me, I found out that the eminentia does not always represent the position of the superior semicircular canal and, due to this and other anatomical anomalies, any mathematical approach will sometimes lead to disaster.

When pus is located anterior to the labyrinth and adequate drainage cannot be obtained by the usually effective method of following "leads" from the mastoid cavity, it then becomes necessary to approach the infected area directly. After studying petrous pyramid specimens of all types, including cadaver material, histological sections and prepared bones cut serially, I came to the conclusion that the superior surface of the pyramid offered greatest possibilities for exposure, visibility and safety. The acoustic apparatus need not be disturbed. The dangerous elements are the possibility of injuring the facial nerve, injury to the carotid artery and its pericarotid venous plexus or laceration of the dura.

Since there is no limitation of an exact nature on the term petrous pyramid, I wish to propose a boundary for description purposes in the presentation. Pass two parallel planes

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through the petrous pyramid, perpendicular to its long axis. Plane A is passed through the anterior extremity of the labyrinthine bony capsule and Plane B is passed through the posterior extremity. The area between these two planes will be named the perilabyrinthine region of the petrous pyramid and the area anterior to Plane A will be termed the antelabyrinthine region. Because there is no natural boundary between the cells of the mastoid and those of the petrous pyramid, some such division seems necessary. With this nomenclature it is obvious that the old term of perilabyrinthitis is accentuated in its importance, since it also signifies an early involvement of the pyramid. It is, so to speak, a port of call on the pathway to the tip.

Our study of petrouisitis has really progressed backwards. Gradenigo's syndrome is accepted as the end-result of a process which in most instances has been an extension from a diseased middle ear and mastoid. All roads must, therefore, lead by the labyrinth. In reviewing our cases, therefore, we are not surprised to find that those of long standing and some of the more fulminating ones complain of dizziness, nausea, tinnitus, instability or, in other words, present objective symptoms of labyrinthine disturbance. The symptoms may have occurred some time previous to the actual onset of recognized petrous symptoms and they may have been so transient and mild as to have been ignored, but they are frequently present. I recall one patient operated by Dr. Moorhead of Brooklyn, during the past year, whom I saw in consultation during her labyrinthine attack some six months previous to the operation. At the time I saw her, the middle ear was dry and apparently the drum was normal. She had a history of a previous otitis which resolved. In view of the fact that at operation a destroyed petrous pyramid was found, it appears logical that the labyrinthine symptoms indicated a period in the progress of the infection. The prodromal symptoms, if the liberty of such a designation is permitted, may be an important bit of evidence in deciding if the antelabyrinthine portion of the petrous pyramid is involved. The classical symptoms of a Gradenigo's syndrome need careful study, since, individually, they may be caused by many other conditions besides pus in the petrous pyramid. The VIth nerve is frequently involved by a lesion in the posterior cranial fossa. Nuclear lesions which have been the result of pres-

sure or degeneration also involve the Vith nucleus. Hemispheres as the accompanying symptom in Gradenigo's syndrome results from so many other lesions of the head that differential diagnosis is difficult. It will be readily seen, therefore, that the mere occurrence of a discharging ear and a concomitant hemispheres, plus external rectus paralysis, may not be diagnosed as petrositis without a thorough neurological examination to detect other possibilities.

A simple observation, but one of practical value, is the behavior of post-operative mastoid discharge. An uncomplicated mastoid operation by which a complete removal of mastoid cells is done should be followed, after a few days, by a gradually diminishing discharge from the aural canal and mastoid wound. When the discharge persists, unabated or increased in amount, even after a few weeks, probably the petrous cells are infected. Realizing the rashness of this statement, I will hasten to call attention to two previously made statements. One, that all cells were removed from the mastoid process at the time of the first operation, and the other, that the perilyabyrinthine cells are anatomically part of the petrous pyramid. Obvious exceptions to this statement will immediately come to mind. Osteomyelitis, tuberculosis, syphilis and malignancy are among the exceptions. The main idea is, nevertheless, sound. It might be advisable, at this point, to mention one, not too uncommon, cause of persistent discharge, and that is a foreign body. Although we have all found pieces of rubber drains, gauze, pledgets of cotton, broken bits of instruments and other oddities, we can hardly confuse the issue by including this accidental group.

When pus is located in the perilyabyrinthine area, dizziness, nausea, headache, instability, nystagmus, are commonly encountered symptoms. Petrositis of the perilyabyrinthine area seems a proper term. These patients are best treated by reoperation and exploration from the field of the former operation. The need for more elaborate operations, either through the pyramid or from without, by penetrating the walls is rare. The total number of reported cases where such procedures have been used bear ample proof of the foregoing statement. That the need of a surgical approach to the antelabyrinthine region of the petrous pyramid does exist also has proof in the fact that cures have resulted from such operations on patients who in all probability would otherwise have

died. The operations of Eagleton, Friesner, Kopetzky, Almour, Ramediez and others have a proven value. Situations arise in which these operations are particularly suitable.

For some time I have believed that the least dangerous and most effective approach would be through the superior surface of the pyramid. Friesner has repeatedly stated that when the infection is in the pyramid or pus has escaped through the superior surface, surgery of the radical mastoidectomy type is not necessary.

While experimenting on a cadaver, Dr. J. Arthur MacLean suggested a change in my original incision to the incision advocated by Dr. Max Peet* for the operation on a Gasserian ganglion. I assisted Dr. MacLean on two cases where he sectioned the ganglion and I was pleased with the complete visualization of the superior surface of the petrous pyramid. The technique employed in this approach to the petrous is the same as the Gasserian ganglion approach. A method of approach to the part of the petrous pyramid which contains the major number of cells is possible by an addition to the Gasserian technique. The main advantage of the Gasserian operative technique is an advance through a sterile field. Any elevation of dura from the infected mastoid area has the ever present danger of a complicating meningitis through an accidentally lacerated dura. This element of danger is almost completely eliminated by the ganglion approach.

When the patient is in a prone position, the intracranial pressure is sufficient to make the retraction of dura and the underlying brain difficult. Dr. Friesner has overcome this difficulty by using ventricular aspiration. According to the Peet method, the patient is placed in a sitting position, which position so reduces intracranial pressure that dural retraction is easy. A full, direct view of the superior surface of the petrous is easily obtained.

Preoperative Preparation: The hair is removed from the operative field. This necessitates a rather more extensive removal than a wide mastoid preparation. The clean area should extend at least one inch above the temporal muscle attachment.

*Dean Lewis' Surgery. Vol. 9, Chapter 2, Max Meier Peet, "Cranial Nerve Surgery."

A preliminary sedative is advisable. Two methods of anesthesia adapt themselves nicely to the requirements, avertin and local anesthesia, or intratracheal cyclopropane. Inhalation of ether is satisfactory.

The usual $3\frac{1}{2}$ per cent of iodine, followed by alcohol, are used as antiseptics.

Operation: A scratch incision is made in a perpendicular direction starting at the attachment of the temporal muscle and extending downwards to the zygomatic process one inch anterior to the external auditory meatus. This line of incision will parallel the fibres of the temporal muscle and the superficial temporal artery. The actual incision should extend through the periosteum to the bony surface of the cranium. The periosteum is then elevated to give as wide an exposure of bone as possible. The lower portion especially should be elevated below the upper border of the zygomatic process. A trephine opening is made with a Hudson bur posterior to the junction of the greater wing of the sphenoid and the squamous portion of the mastoid just below the parietal bone. From this opening the necessary amount of calvarium is removed by rongeurs. This removal will roughly include part of the greater wing of the sphenoid, the parietal bone lying below the temporal muscle attachment, and the anterior two-thirds of the squamous portion of the temporal bone. The elevation of dura along the floor of the middle fossa is the next procedure. Most of the dura will elevate easily by means of cotton pledgets, suction tips, blunt dissectors, etc. At some places the dura is adherent and in these areas care must be used. The most common places where adherent dura may be expected are in the vicinity of the epiantral or epi-tympanic regions, over the geniculate ganglion, at the exit of the greater and lesser superior petrosal nerves, and around the middle meningeal artery. The first and most important structure to locate is the middle meningeal artery. Peet advocates a little wooden peg placed in the foramen spinosum to control bleeding from this vessel. Cotton pledgets are adequate. The upper portion can be grasped with a brain clamp and as a double precaution this clamp may be touched with a desiccating electric current. The artery is then severed to allow further elevation of the dura. Proceeding forward and mesially from the middle meningeal artery the next structure encountered will be the third division of the Vth nerve as it

passes from the ganglion through the foramen ovale. The usual location is mesial and anterior to the foramen spinosum. Posterior and mesial to the middle meningeal artery the grooves housing the superficial petrosal nerves will be found. At the posterior limit of these nerves will be the hiatus Fallopii from which the greater superficial petrosal nerve emerges. The danger of a facial paralysis following traction on this nerve has been mentioned by Peet. His explanation is that the traction disturbs or injures the geniculate ganglion. Being freed of the foregoing structures, the elevation of dura progresses below the Gasserian ganglion and over the Gasserian fossa. The superior petrosal ridge is now easily identified. In the examinations I have made so far I have not found true bone over the carotid artery in the area of the Gasserian fossa. Most textbooks state that the carotid artery is covered by fibrocartilaginous tissue. The aperture is the foramen lacerum medium. It is therefore easy to locate the bony edge of the foramen and, by means of a small right angle elevator, curette and biting forceps (sphenoid forceps) remove the roof in a posteromesial direction. This area contains the major cellular portion of the petrous pyramid as demonstrated by the gross dissections and microscopic specimens. Downward pressure on the carotid artery may be necessary after its exposure in order to permit complete removal of the cells comprising that portion of the petrous pyramid adjacent to the posteromesial wall. This procedure, therefore, exposes that portion of the pyramid between the internal auditory meatus and the tip.

Provided the infected area has been found, two methods of drainage are possible. Either posterior through the mastoid wound or laterally through the avenue of approach. Where extensive procedures have been done on the mastoid, the two fields are easily connected as shown by the illustration. Provided no infection is found, the field, being sterile, may be closed.

Any operative procedure in which there is danger of injury to a major blood vessel must include steps for control of hemorrhage. In this operation there is the possibility of injuring both the internal carotid artery or the pericarotid plexus. Since these structures are encased in a bony canal, pressure should control the immediate hemorrhage and the possibility of internal carotid ligation in the neck should be kept in mind.

We have specimens procured at autopsy which show pus bathing the carotid in its course through the petrous pyramid and in these specimens there is also found a thrombosis of the pericarotid venous plexus. We have, in the surgical procedure just described, the following pathological lesions which may be reached.

1. Extradural abscess in the middle fossa which has resulted from an exit of pus from the petrous pyramid.
 2. Extradural abscess in the posterior fossa which also had its origin in the petrous pyramid, but ruptured through the posteromesial wall.
 3. Empyema of the antelabyrinthine portion of the petrous pyramid.
 4. Pericarotid abscess with or without venous thrombosis.
 5. A speculative attack on cavernous sinus thrombosis.
- 121 East 60th Street.

SARCOMA OF THE LARYNX.

DR. ISIDORE ARONS, New York.

Of the 60-odd cases of malignancy of the larynx and pharynx which I have treated during the past 10 years, several were classified as reticular-cell sarcoma of the lymphoid tissue, while only one was lymphosarcoma. A survey of the literature reveals that lymphosarcoma of the larynx is relatively rare, occurring in about 0.5 per cent of all malignancies of the larynx. We, therefore, report the following case:

CASE HISTORY.

J. C., male, age 60 years, first consulted me, Sept. 26, 1935, because of increasing hoarseness, noticing that he began to lose his voice about a year ago. The patient was in charge of a group of laborers and had to shout and use his voice a great deal. Five weeks prior to his visit in my office, the patient had caught a "cold." Since then he had had difficulty in breathing and wheezes a great deal. He had not lost any weight and ate well.

Laryngeal examination revealed an edema on the left side, which extended to and closed the ventricle of the larynx. The cords were not visible. External palpation revealed an infiltration about 1.5 cm. in thickness at the external lateral position of the larynx on the left side, but no palpable glands.

The muscle attached to the right side of the larynx was movable, though thickened, suggestive of an invasion by the neoplasm. The larynx was movable in its upper portion but fixed in its lower portion. This condition appeared to be intrinsic and extrinsic sarcoma of the larynx (see Fig. 1).

A biopsy by Dr. Scheer on Sept. 24, 1935, at Polyclinic Hospital was interpreted by Dr. Preis: "Microscopical examination shows several small portions of tissue from the lower larynx, composed chiefly of mucous membrane with some lymphoid tissue beneath it. One portion appears somewhat polypoid in character. The tissue is edematous, vascular, congested and, in some areas, presents a definite inflammatory lesion. There is no evidence of carcinoma. The lymph nodules found immediately beneath the epithelium show changes which can be explained on an inflammatory basis. But in one section a very thin margin of the tumor has been touched, and this small area consists of atypical densely stained and closely packed lymphocytes with little cytoplasm in a definitely abnormal arrangement. This justifies the opinion that lymphosarcoma is present."

Diagnosis: Lymphosarcoma (see Fig. 2.).

TREATMENT.

A cycle of 25 X-ray treatments was outlined; daily treatments, 180 kv., 4 ma., 60 cm. distance, 1 cu. and 2 al. filtration; 10 x 10 cm. fields, alternating right and left lateral necks. The first series of treatments

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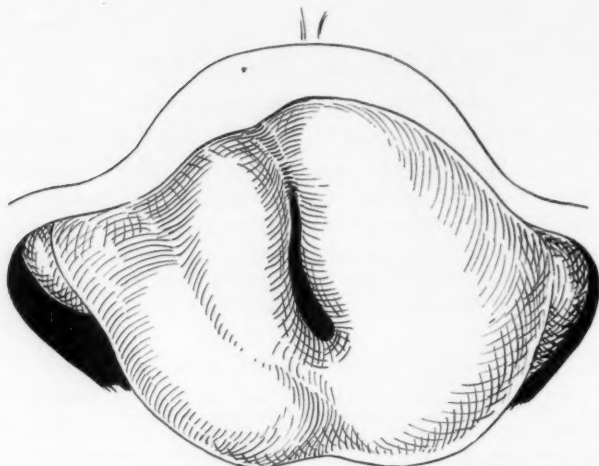


Fig. 1. Case 7,295. J. C., age 60 years. Sept., 1935. Intrinsic and extrinsic neoplasm. Biopsy: Lymphosarcoma of larynx. Before treatment.

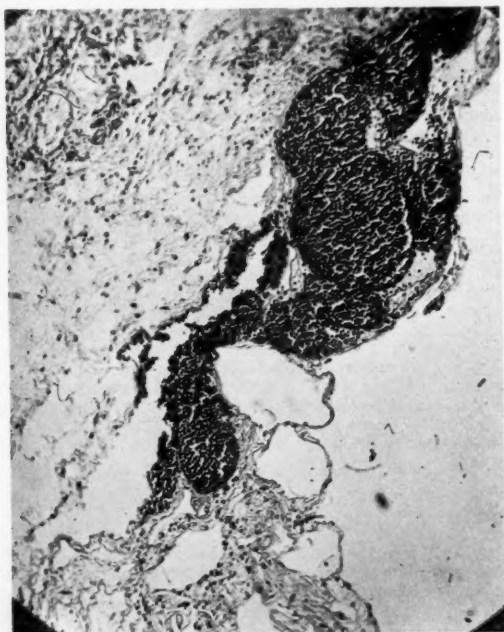


Fig. 2. Microphoto of biopsy (sarcoma of larynx).

200 Roentgens* each to each side of the neck and the last 10 treatments of 150 Roentgens each were given within a period of four weeks, each area receiving a total dose of approximately 3,000 Roentgens. This cycle was combined with ultra short wave therapy.

When seen about six weeks after completion of this cycle, the patient had gained eight pounds and had regained his voice. No definite infiltration of the larynx was visible. The patient was discharged and advised to report to Dr. Scheer for monthly check-up. The infiltration on the left side had almost completely subsided, leaving the left arytenoid visible (see Fig. 3).

A second cycle of X-ray treatments to the right lateral neck was given from June 29 to Aug. 19, 1936, with the same factors as previously. The patient received 150 Roentgens per treatment three times weekly, with the intention of increasing the doses to a total dose of 2,700 Roentgens.

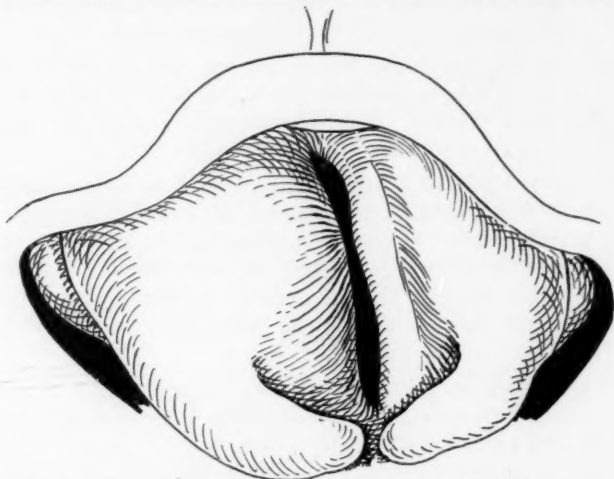


Fig. 3. After treatment. Oct., 1935. Patient well. Free from symptoms. June, 1936. Patient well. No recurrence.

This cycle was interrupted on July 7, when a tracheotomy was performed by Dr. Scheer at Polyclinic Hospital because of the reaction. Treatment was resumed on July 22, 1933.

Examination at the end of the cycle revealed the condition to be under control. A re-examination in October showed that a few additional treatments were indicated and it was suggested that the patient have an ebonite tube inserted for further irradiation. He refused this, as well as any further treatment or operative measure. His family reported his death in January, 1937, one and one-half years after his first appearance at my office, and probably two and one-half years since the initial onset of his illness.

A more favorable end-result would probably have followed had a tracheotomy been done on this patient before any X-ray irradiation had been started. This suggestion, however, was refused by the patient.

The present method of treatment which I employ in this type of case is based upon Coutard's technique. Tumors of the larynx and pharynx are irradiated with single, small doses and small hour intensity

*Measured in air.

with a high total dose, administered over a long period of time. Contrary to the classical Roentgen treatment, there is a definite skin injury, which usually disappears in four to six weeks.

EFFECTS AND COMPLICATIONS.

Soreness of the throat, dryness of the mouth and difficulty in swallowing are present during the first week of treatment, as well as the beginning of the white membrane. During the second and third weeks, there is an extension of the white membrane and the saliva becomes stringy, with the result that it is difficult to clear the throat. This change in the mucous membrane, known as radioepithelitis, heals within two weeks, leaving a practically normal mucous membrane.

Towards the end of the treatment cycle the irradiated skin becomes dark red, the surface peels off, leaving large moist patches. This change is called radioepidermitis and takes longer to heal — about six to eight weeks.

During the course of these treatments there are certain difficulties which cause complications. Most patients complain of a sticky mucus during and immediately following the mucous membrane reaction. Dryness of the mouth from the lack of saliva persists for some months. There is also a disappearance of the sense of taste, which, in some instances, returns within a few months.

All the above complications are unpleasant to the patient, but he should be told that in most cases these are only temporary, and are a result of the treatment.

CONCLUSIONS.

A survey of the literature does not permit any definite conclusions regarding the prognosis of cases of lymphosarcoma of the larynx, depending upon the extent of the lymphatic involvement at the onset of treatment. We believe that the protracted, fractionated treatment method is indicated, keeping the lethal dose within the lower limits of the range and delivering this dose within a period of three to four weeks. This results, at least, in palliation and the prolongation of life under comparatively comfortable circumstances.

I am indebted to Dr. Henry Scheer for his permission to report this case.

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**TUBERCULOUS TONSILLITIS: A CLINICAL-
PATHOLOGIC STUDY BASED ON
782 TONSILLECTOMIES.***

DR. J. H. AHRONHEIM, Jackson, Mich.

The problem of the tuberculous tonsil deals with three points which are of pathological, clinical and social interest.

1. The percentage of tuberculosis in tonsils.
2. The possible tuberculous involvement of distant organs, the lesions in the tonsils being either secondary or primary.
3. The importance of a more or less extensive tuberculosis of the tonsils to the patient himself.

Point one requires routine histologic examination of a large material of tonsils, selected at random from the general population, and omitting sources which are apt to supply a greater tuberculous material than the average general hospital. Point two will be answered by a very careful physical examination of the patient; whether the local tonsillar tuberculosis is primary or secondary can be decided in many cases by extent and histologic appearance of the lesion. Point three requires examination of the patient and observation over an extended period of time.

PREVIOUS INVESTIGATIONS.

A great number of investigators have approached the problem of tuberculous tonsillitis from various angles, quite a few reports dating back to the latter part of the last century. Most of these investigations deal essentially with the percentage of the tubercular-positive tonsillectomies. A comparison of the results obtained by a number of authors (see Table I) demonstrates a remarkable difference of the various figures, which range from 0.44 per cent up to 9 per cent.

Undoubtedly, type and size of material must be made, in part, responsible for the great variation of the figures presented. In some instances the material is too small; in others,

*From the Pathologic Laboratories of the W. A. Foote Memorial and Mercy Hospitals, Jackson, Mich.

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the source of the material is such that it does not guarantee an at-random selection from the general population. But a comparison of the figures, representing the percentage of tuberculous tonsillitis, with the years of publication shows a very distinct decrease in the incidence of tuberculosis, as already mentioned by Wilkinson,⁹ in 1929, and Magee,¹² in 1937. A merely coincidental trend seems highly unlikely if Magee's recent and Weller's⁵ report of 1921 are compared, both of which are based on an unusually large material and were obtained in the same institution.

The study of the relationship between tuberculosis of the tonsils and the entire body shows that in many cases of

Table I.
Percentage of Tuberculous Tonsillitis as Reported in the Literature.

Investigators	Number of Cases	Tb. Tonsils Percentage	Year of Pub.
Wood ¹ (quoting 23 authors)	1,671	5.2	1905
Sewall ²	772	6.2	1911
Mitchell ³	100	9	1916
Crowe, et al. ⁴	1,000	4.6	1917
Weller ⁵	8,697	2.35	1921
Mullin ⁶	400	4.25	1923
MacCready and Crowe ⁷	3,260	4.18	1924
Howarth and Gloyne ⁸	95	5	1924
Wilkinson ⁹	10,000	0.52	1929
Rossner ¹⁰	296	1.01	1933
Newhardt, et al. ¹¹ (own material)	100	1	1934
(Quoting literature)	30,676	2.03	1934
Magee ¹²	6,359	0.44	1937

advanced pulmonary tuberculosis there is also an involvement of the tonsils. Mullin's⁶ material showed that 60 per cent of patients with tuberculous tonsils had a positive history of tuberculosis; of these, 70 per cent had bilateral pulmonary tuberculosis. G. B. Wood¹ quotes seven authors, who found 94 cases of tuberculosis of the tonsils in 136 cases of pulmonary tuberculosis. Newhardt and co-workers¹¹ examined the tonsils of 71 patients with positive sputum and found tuberculous involvement 41 times. Heaton¹³ collected 80 cases of tonsillectomies from sanatoriums and tubercular resorts; he found 13 cases of tonsillar tuberculosis, of which six had advanced, six moderately advanced, and one minimal pulmonary tuberculosis. Otto's¹⁴ autopsy material of 45 patients who died of tuberculosis revealed involvement of the tonsils

in 74 per cent. Strassmann¹⁵ reports 13 cases of tuberculous tonsillitis in 21 autopsies on tuberculous patients. Fischer¹⁶ found the tonsils involved 106 times in 161 autopsies on patients with pulmonary tuberculosis. While most of these investigators believe in a secondary infection of the tonsils, probably through infected sputum, Hudson and Wollaston¹⁷ suggest a secondary involvement of the lungs from a primary tuberculosis of the tonsils. Similarly, Van Zwaluwenburg and Grabfield¹⁸ interpret the "apical cap," a pleural thickening over the apex found by X-ray, as a possible secondary involvement originating in the tonsils. They found that out of 16 cases of tuberculous tonsils, 15 showed this apical cap, while it was found in only three cases out of 27 who were known to have no tubercles in the tonsils. A definite relationship between enlarged cervical glands and tuberculous tonsillitis is emphasized by Mitchell,⁹ who found tuberculous tonsils in 38 per cent of cases with enlarged cervical glands. In 98 cases of lymphnodes suspicious of tuberculosis, Schlittler¹⁹ found the tonsils involved by tuberculosis 48 times. Realizing that there is a direct extension of infection from the tonsils to the cervical lymphnodes, Howarth and Gloyne⁸ suggest tonsillectomy rather than adenectomy in cases of tuberculous lymphadenitis.

DISCUSSION OF PRESENT MATERIAL.

The present material was obtained from tonsillectomies performed at the W. A. Foote Memorial and Mercy Hospitals, Jackson, Mich. The specimens were sent to the pathological laboratories in 10 per cent formalin. Routinely, cross-sections about 3 mm. in thickness were cut out of the centre of each tonsil and, if available, a suitable piece of adenoid added. The tissues were embedded in paraffin and the sections stained with hematoxylin-eosin according to the standard procedures.

Tuberculosis was discovered in six out of 782 pairs of tonsils, yielding a percentage of 0.77. It must be realized, however, that this figure is probably not correct in regard to the actual percentage of tuberculous tonsillitis. Serial sections of the six tuberculous tonsil pairs revealed that, in three cases, the tuberculous lesions were found only in one level of one tonsil. The discovery of tuberculosis in these three cases was, therefore, more or less coincidental. It even must be assumed that a number of tonsils which were recorded as nontubercu-

Later studies

TABLE II.
Clinical Information on Six Cases of Tuberculous Tonsillitis.

Pat. Age, Sex	Symptoms	Tb. in Family	Tb. Habitus	Cervical Glands Before Operation	Weight Following Operation	X-ray	Skin Test
1. M. F., 16, F.	Frequent sore throats	Yes	No	Slightly enlarged	No changes	Negative	Negative
2. F. P., 20, F.	Frequent sore throats	No	Yes	Enlarged	No changes	Negative	Negative
3. C. K., 27, M.	Frequent sore throats	No	No	Enlarged	Gained 6 lbs.	Old, inact. pulm. tb.	Not recorded
4. M. G., 23, M.	Progressive deafness	No, but tb. contact	No	Not enlarged	Lost 5 lbs.	Negative	Negative
5. C. D., 14, M.	Cough, weak, night sweats	No	Yes	Enlarged	Gained 11 lbs.	Negative	Negative
6. S. D., 32, F.	Frequent sore throats	No	No	Not enlarged	Gained 12 lbs.	Refused	Refused

lous did contain lesions in levels not examined. Newhardt and his co-workers¹¹ obtained serial sections from blocks of 20 tonsil pairs which had been diagnosed tubercular-negative. They found tubercles in the additional sections of four of these cases. In a similar procedure, Sewall² discovered tuberculosis in one out of 20 tonsils which originally were believed to be free of tubercles.

The ages of the six cases of tuberculous tonsillitis ranged between 14 and 32 years. This coincides with Magee's¹² statement, who found the greatest incidence of tuberculous tonsils in the age group of 21 to 30 years, while the lowest was found in the youngest group. Crowe and his co-workers'⁴ material of 46 cases of tuberculous tonsillitis is distributed as follows: four to five years, five cases; five to 10 years, 14 cases; 11 to 15 years, 10 cases; 16 to 25 years, 13 cases; over 26 years, four cases.

In regard to sex, the present material shows no preference, both sexes being represented three times. Weller,⁵ in his considerably larger material, found a slightly greater incidence in females.

The six patients in whom tuberculous tonsillitis was found were subjected to a careful physical check-up, including chest-plate and skin test for tuberculosis (Mantoux). The last reports on the patients' condition were obtained between six and 11 months following tonsillectomy.

The preceding table, summarizing briefly the important clinical data on all six patients, shows that none of them presents a characteristic picture suggesting tuberculosis. Only in one instance the symptoms indicating the operation were suspicious of a possible pulmonary lesion. One out of five chestplates revealed an old, inactive focus. Loss of weight following operation was recorded once. None of the four skin tests available were positive for tuberculosis. The most recent information obtained from the patients revealed that all seemed to be in perfect health.

The actual physical condition of patients with tuberculous tonsils is by no means sufficiently revealed by clinical data only. It is imperative to study even the most favorable clinical picture in conjunction with the histologic appearance of the tuberculous lesions.

Table III, presenting a brief histological description of the six cases of tuberculous tonsillitis, shows that three of these six cases contain only a single unilateral lesion, two show miliary, and one multiple caseating confluent tubercles.

PATHOLOGIC INTERPRETATION.

A correct pathologic interpretation of these lesions requires the determination whether they were primary or secondary in the tonsils.

We are dealing with a primary tonsillar tuberculosis if the tubercle bacilli are lodged in the tonsils without having passed

TABLE III.
Histological Description of Six Cases of Tuberculous Tonsillitis.

Pat.	Histology of Lesion	Extent of Lesion	Pathological Interpretation	Prognosis
1. M. F.	Several confl. cas. tubercles	Unilat.; only one level	Primary tuberculosis	Excellent
2. F. P.	Miliary tubercles confined to germ centres	Bilat.; lesions in all levels	Secondary tuberculosis	Doubtful
3. C. K.	Two tubercles. Also occasional mil. tubercles in germ centres	Bilat.; lesions in sev. levels	Secondary tuberculosis	Doubtful
4. M. G.	Small lesion of sev. tubercles	Unilat.; only one level	Primary tuberculosis	Excellent
5. C. D.	Single medium-sized lesion	Unilat.; only lesion found	Primary tuberculosis	Excellent
6. S. D.	Multiple cas. tubercles	Bilat.; lesions in most levels	No interpretation	Doubtful

through other organs. This infection may take place through ingestion (Mitchell,³ Heaton¹³) or inhalation (Weller⁵). In both instances, the infection is mucosal in type; the lesion is usually unilateral and may be found in only one level of the block. Secondary tonsillar tuberculosis may develop from infected sputum in cases of open pulmonary tuberculosis, or as a blood stream infection, quite commonly in tuberculosis of the mediastinal lymphnodes. In the former instance, a picture develops similar to a primary tonsillar lesion, the infection being mucosal in origin. In the latter instance, a miliary spread is found, which is always bilateral and, according to Weller,⁵ involves most commonly the germ centres of the tonsils only. Etiologically, Krauspe²⁰ classes these two

distinctly defined groups as resorptive and hematogenous, and demonstrated them experimentally on rabbits by local application and injection of tubercle bacilli, respectively.

Applying these considerations to the six cases of tonsillar tuberculosis presented, the following conclusions can be drawn. Cases 1, 4 and 5 are most probably cases of primary involvement. Cases 2 and 3 must be regarded as secondary, although the possibility that the primary focus was also in the tonsil cannot be excluded. In case 6, in which the clinical

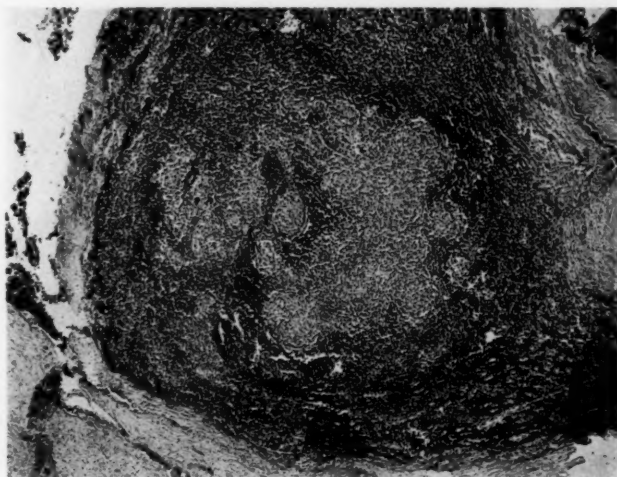


Fig. 1. Several confluent epithelioid tubercles of the tonsil. Mucosal type of infection (Case 1). Low power.

information through the unco-operative attitude of the patient was insufficient, a pathologic interpretation is impossible. The infection, which in this case is apparently mucosal in type, may have taken place primarily from the outside. The bilateral and very extensive involvement, however, suggests more a secondary infection, which can only be excluded by X-ray examination of the chest.

PROGNOSIS AND SIGNIFICANCE.

In all cases of tuberculous tonsillitis, no matter whether primary or secondary, it is safest to keep the patient under

observation for some time. The prognosis of primary tuberculosis without any symptoms of tuberculosis elsewhere in the body developing within several months following tonsillectomy, is undoubtedly excellent; in these cases it must be assumed that the tonsillar lesion was the only focus which was eliminated through tonsillectomy. Cases of tuberculous tonsillitis which are interpreted as secondary have to be considered doubtful in their prognosis until a long period of observation has proven that the primary tuberculous focus,

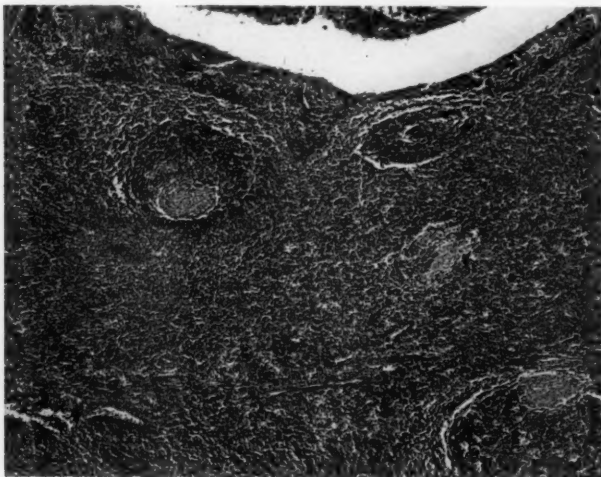


Fig. 2. Miliary tubercles of the tonsil confined to the germ centres. Hematogenous type of infection (Case 2).

which, with all probability, is still existing in the body, is definitely quiescent and inactive. MacCready and Crowe⁷ report the outcome of 50 patients, in whom tuberculous tonsillitis had been diagnosed; 40 were observed over five to 10 years, 10 over at least two years. Development of tuberculosis of the cervical lymphnodes was found twice; of the mediastinal glands, three times; five patients developed pulmonary tuberculosis; two, tuberculosis of bones and joints. The authors do not state, however, whether they considered these cases as primary or secondary tonsillar tuberculosis so that definite conclusions do not seem justified from these observations.

The significance of a tuberculous involvement of the tonsils to the patient is apparently somewhat under-estimated. As early as 1884, Strassmann¹⁵ and, more recently, V. V. Wood,²¹ Mullin,⁶ McCready⁷ and others denied the tuberculous tonsil of any clinical importance.

Unquestionably, a secondary tonsillar tuberculosis in a case of known pulmonary tuberculosis is an entirely insignificant finding. But if a tonsillar lesion, found in routine examination, leads to the detection of an active tuberculosis elsewhere in

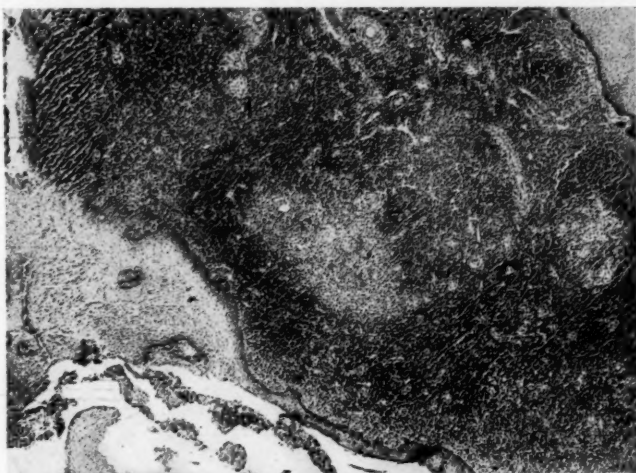


Fig. 3. Multiple caseating tubercles. Mucosal type of infection (Case 6). Low power.

the body, the significance of this finding is obvious. Even a primary tonsillar involvement is of importance as a warning signal of a possible extension of this lesion into neighboring or distant parts before its removal. The question whether a routine examination of all tonsils and adenoids should be carried out should, therefore, be answered in the affirmative. Even the small number of positive cases detected is unquestionably worth the seemingly unnecessary efforts of routine examinations.

SUMMARY.

Six cases of tuberculous tonsillitis were found in a material of 782 tonsillectomies, giving a percentage of 0.77. Clinically,

these six cases showed practically no evidences of an active tuberculosis. Three were believed to be cases of primary, two of secondary tonsillar tuberculosis; one case was doubtful.

The prognosis of tuberculous tonsillitis depends on both clinical observation of the patient and correct pathologic interpretation of the lesion. The significance of a tubercular-positive finding in one or both tonsils may be little in some cases; but, as it may lead to the discovery of an active tuberculous process elsewhere in the body in other cases, routine histologic examination of the tonsils seems justified.

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THE CROOKED NOSE.*†

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This topic presents many interesting features. The chief concern is either a nasal obstruction or the apparent external deformity. It predominates more in the male than in the female. This is because of the traumatic element. It is observed in all ages.

The etiologic factor in all cases examined was invariably trauma. The traumatic injury sustained was either inflammatory or noninflammatory in type. The deviation in the inflammatory type was due to the subsequent cicatrization. The underlying factor in the noninflammatory type was the fracture or dislocation or fracture dislocation of the septi nasi.

For descriptive purposes, let us divide the external nose into thirds. The anatomy involved in the upper third consists of the nasal bones, the frontal processes of the maxillae and the perpendicular plate of the ethmoid. Any undue trauma in this region can readily cause a fracture or dislocation, or both, to the perpendicular plate of the ethmoid, as well as the nasal bones. This results in the deviation of the upper third of the nose in the direction of the dislocation. Fig. 1. illustrates this type of deviation, and Fig. 2 shows the correction.

The late Joseph, of Berlin, attacked this problem by removing a triangular segment from the broad aspect of the deviation. This technique is not only difficult but wholly unnecessary. A more simplified approach would be a high resection of the fracture dislocation of the bony septum. The nose is then narrowed in the usual plastic procedure by severing completely the nasal bones from their lateral bony attachments and by digital manipulation, the upper third of the nose can easily be brought directly in midline. When the severed nasal bones fail to remain in midline it is invariably due to some tension in the remaining bony septum. Any strong, straight, blunt instrument will serve to release this tension by exerting pressure internally against the dislocated segment.

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The middle third of the nose is formed by the quadrilateral septal cartilage and the upper lateral cartilages. Unilateral hypertrophy of the upper lateral cartilage or cicatrization following a septal abscess is at times the background for the deviation in this region; however, the deviation in this region is chiefly due to a fracture dislocation of the quadrilateral cartilage of the septi nasi following traumatic injury.

The corrective measures naturally depend upon the pathology present. In the unilateral hypertrophy of the upper lateral cartilage, the deviation is apparent, but not real. Removal of the excess segment of the hypertrophied cartilage usually suffices. In the inflammatory type, where cicatrization and synechiae are present, complete eradication of the visible



Fig. 1.



Fig. 2.

pathology is very often insufficient. The contracting forces of the fibrosis have in these cases caused a bowing of the quadrilateral cartilage, which, in turn, accounts for the deviation. The tension in the cartilage is released by blunt instrumentation. When the fracture dislocation of the septal cartilage is at fault, a complete submucous resection is invariably sufficient. Fig. 3 illustrates this type of deformity, and Fig. 4 shows the correction.

The lower third, or the tip of the nose, is by far the most difficult to handle. The factors that come into play here are the alar cartilages with their lateral and medial crura, the columella, and the anterior portion of the septum. The most important factor observed was the faulty attachment of the

septum to the anterior nasal spine, which accounted for the real deviation of the tip of the nose in practically all our cases. Normally, the septum is attached in a groove in the centre of the anterior nasal spine. In the deviated tip, the septum was attached to the lateral aspect of the spine, which caused a torsion of the anterior part of the septum, and this, in turn, caused the deviation. Figs. 5 and 6 show this deformity, with correction. The problem here is to sever this faulty attachment, release the created torsion and overcorrect the deviation. The following modified technique, which has proven very efficacious in these difficult problems, is presented.

Fig. 7 shows the first view, exposing the anterior portion of septum free from its perichondrium and its faulty attach-



Fig. 3.

Fig. 4.

ment to the anterior nasal spine. View 2 shows a chisel separating the faulty attachment of septum to the nasal spine. The separation continues along the base of the nose, depending upon the extent of the deformity. View 3 shows scissors cutting the septum in midportion at right angles. The purpose of this is to release the tension in the septum created by its faulty attachment to the nasal spine. View 4 represents a top view, showing the angle of the needle going through the septum. The tip is deviated to the left. The needle on the right side goes through the septum somewhat posteriorly and comes forward anteriorly on the left side of the septum. Fig. 8, the fifth view, shows the needle passing through the columella at the same level of the septum. The mark "X"

represents where the needle came through from the other side of the septum. View 6 shows the completion of the three columella septal mattress sutures. When these sutures are tied, the tip of the nose will become overcorrected to the right



Fig. 5.



Fig. 6.

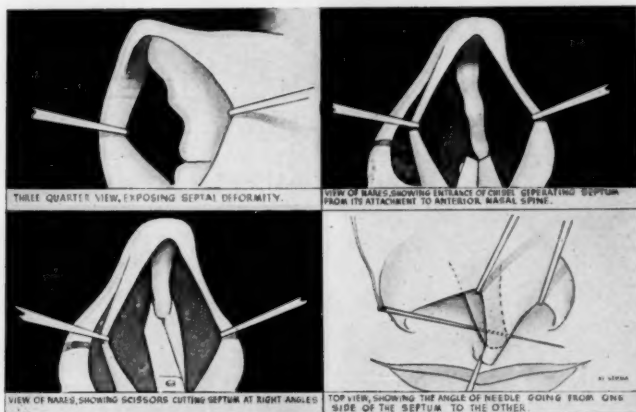


Fig. 7.

side. This is further reinforced by the application of adhesive, as shown in Views 7 and 8. The relative position of adhesive is very important. The short end of adhesive is placed on the left side, as seen in View 8, and attached with its long end to the right side. The pull created by the long ends of

the adhesive will tend to keep the tip in the overcorrected position. Sutures and adhesive are removed in seven days. Gentle digital manipulation will bring the tip back in the midline, where it will remain permanently.

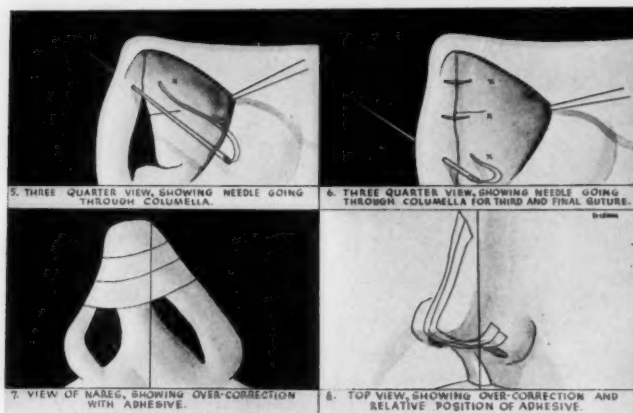


Fig. 8.



Fig. 9.

Fig. 10.

The deviated nose may present any of the above deformities or any combinations. Figs. 9 and 10 represent the combined three types of deviation and correction. Naturally, the problem here is to correct all the pathology present, utilizing any or all of the above procedures.

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